



SPECIAL SURGERY

Mohammed El-Matary

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General Surgery

Volume II

Concise Summary Of General surgery



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UPDATED!

Preface

This book provides an update for medical students who need to keep abreast of recent developments. I hope also will be useful for those preparing for postgraduate examination.

This book is designed to provide a concise summary of General surgery, which medical students and others can use as study guide by itself or with readings in current textbooks, monographs, and reviews.

The author is extremely grateful to all the contributors for the high standard of the new chapters, and hopes that you the reader will enjoy going through these pages as much as he had.

M. EI-Matary

Acknowledgement

I'd like to thank who has worked to let this
version of the book comes to light

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Fractures and dislocations general principles

Cortical bone	Cancellous bone
Forms the shafts of long bones	Forms the vertebrae & the ends of long bones
Metabolic turnover is slow	Metabolic turnover is rapid
It can withstand torsional and bending stresses (contributes to rigidity of bone)	It can withstand compression (contributes to pliability of bone)

Blood supply of bones

A) Long bones, is derived from:

1. Nutrient artery:

- Enters the shaft through the nutrient foramen then runs obliquely through the cortex
- Divides into ascending & descending branches in medullary cavity
- Terminates in the adult metaphysis by anastomosing with the epiphyseal, metaphyseal and periosteal arteries.
- Supplies the medullary cavity, inner 2/3 of cortex & metaphysis

2. Periosteal arteries:

- Numerous beneath the muscular and ligamentous attachments.
- Branch beneath the periosteum → supply outer 1/3 of the cortex.

3. Epiphyseal arteries: derived from periarticular vascular arcades.

4. Metaphyseal arteries:

- Derived from the neighbouring systemic vessels.
- Before epiphyseal fusion, the metaphysis is richly supplied with blood by end arteries.



B) Short long bones, (e.g., metacarpals and metatarsals)

- The nutrient artery breaks up into a plexus immediately upon reaching the medullary cavity.

Etiology of fractures

A) Traumatic fractures

1. Direct trauma

- E.g., fracture of the tibia if the bumper of a motor car strikes the bone.
- The fracture occurs at the point of impact.
- In the leg or forearm, if both bones involved → fractured at same level.

2. Indirect trauma

- E.g., i) twisting of the leg → fractures the tibia
- ii) Compression applied vertically on head or feet → fracture the spine

B) Pathological fractures (bones have abnormal fragility)

- Trivial trauma → break

- The causes might be:

1. Local bone diseases as: osteomyelitis, cysts, primary tumours or secondaries

- **Definition of fracture:** structural break in the normal continuity of a bone.

- **Classification of bones:**

a) According to shape

1. Long bones (femur)
2. Short long bones (carpal & tarsal bones)
3. Flat bones (sternum, ribs, and scapula)
4. Irregular bones (vertebrae & hip bone)

b) According to mechanism of ossification

1. Membranous
2. Cartilaginous
3. Membrane-cartilaginous

c) According to maturity

1. Immature (woven) bone
"Collagen is laid down irregularly"
2. Mature (lamellar) bone
"Collagen fibres are arranged parallel to each other" and it has 2 types:

- i) Compact (cortical) bone
 - ii) Cancellous (trabecular) bone → more porous
- The metaphysis is the zone of active growth

2. Generalized bone diseases as: osteogenesis imperfecta, osteoporosis, hyperparathyroidism, Paget's disease and multiple myelomatosis.
(These diseases may lead to multiple fractures)

C) Stress (fatigue) fractures

- They occur secondary to repeated minor loads applied to the skeleton
- E.g., march fractures which occur in 2nd & 3rd metatarsals due to prolonged walking

Clinical feature and diagnosis

A) History of trauma

B) Symptoms:

1. Local pain: ranges from mild to severe.
2. Swelling
3. Loss of function of injured part (varies from minimal to complete)

C) Signs:

Inspection → 1- Swelling & ecchymosis

Due to effusion of blood from broken bone ends & torn soft tissues

2-Deformity due to displacement of bone fragments.

Palpation:

1. Tenderness over a localized point on the surface of the bone.
2. Crepitus when the bone ends are moved against each other.
3. Abnormal mobility (except with incomplete & impacted fractures)

D) Radiological diagnosis

- Radiographs of whole bone including joints in which it participates
- X-rays obtained in at least 2 planes (antero-posterior and lateral)
- In certain circumstances, other views may also be required.

Fracture description

1-Site of the fracture

Intra-articular, epiphyseal, metaphyseal or diaphyseal

2- Extent of the fracture

- a) Complete fractures: the bone is broken into 2 or more fragments
→ tend to displace with loss of opposition & alignment
- b) Incomplete fractures: the bone is incompletely broken → no displacement occurs, as in fissures & greenstick fractures of children

3-Fracture line

- a) Transverse: due to direct violence.
- b) Oblique or spiral: due to indirect violence.
- c) Comminuted: due to severe compression → >2 bone fragments
- d) Avulsion: separation of a bony process with its attached muscles.
- d) Epiphyseal separation in children.

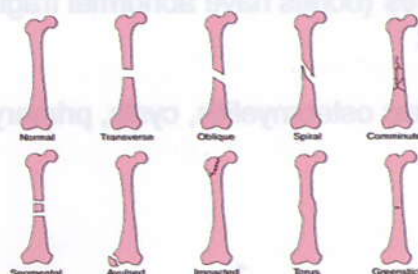


- No history of trauma in stress fractures & some cases of pathological fractures

- Never intentionally look for crepitus & abnormal mobility as:

1. Their confirmation adds nothing to the diagnosis
2. Causes pain & may result in further soft tissue damage

- Examine the motor & sensory innervation of a limb and its circulation, for a complete clinical diagnosis of a fracture



4-Displacement

- Describes the position of the distal fragment in relation to the proximal one, the possible types are:

1. Lateral displacement → distal fragment deviates to one side with loss of opposition.

2. Angulation → loss of the normal longitudinal axis of the shaft.
-It may occur in 1 of 4 directions (anterior, posterior, medial & lateral)

3. Over-riding → distal fragment overlaps the proximal fragment with shortening of the limb.

4. Rotation → The distal fragment is rotated along its long axis

5. Distraction → fragments are separated either by:

- Over vigorous traction during treatment (more commonly)
- Excessive muscle pulls at the time of injury (rare)

6. Impaction.

5-Stability

1- Stable fracture: further displacement after reduction is unlikely.

2- Unstable fracture: displacement is likely to occur as in Oblique, spiral and comminuted fractures.

6- Skin damage (practically the most important description)

1- Simple (closed) fractures: The skin surface is intact.

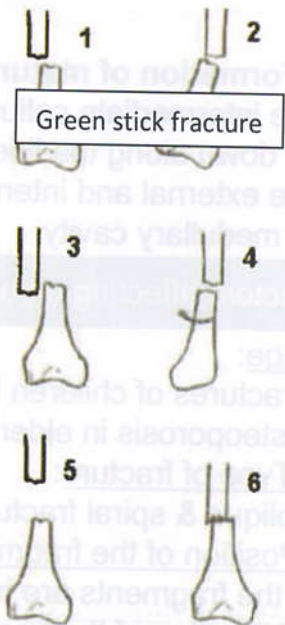
2- Compound (open) fractures: A laceration in the skin communicates with the fracture haematoma → bacteria can reach the fracture site.

-The skin wound may be caused by:

i) Fracture fragment penetrating the skin from its deep surface

"**compound from within**" or

ii) By direct injury to the skin "**compound from without**"



- The chances of significant bacterial contamination is greater in case it is compound from without.
-Fractures of tibia & mandible are commonly compound

Fracture healing

Phases

1. Repair by granulation tissue (lasts for a few weeks)

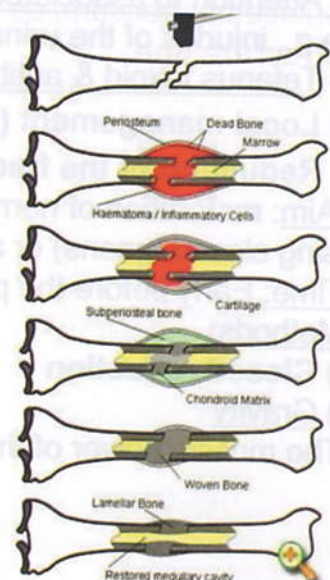
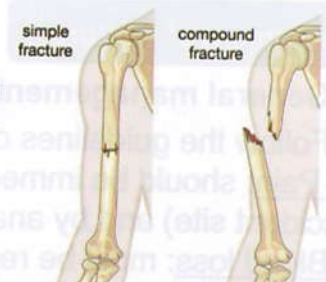
- A haematoma forms between the bone ends underneath the raised periosteum which become invaded by vascular granulation tissue and bridges the fracture gap

2. Union by primary callus (ends at 2-3 months)

- Trabeculae of cartilage invade & replace the granulation tissue.

- Bone cells & matrix gradually appear → formation of an irregular mass of vascular bone and calcified cartilage at the site of the fracture (primary callus)

It first appears on the outer aspect of the fragments (external callus), then along the medullary canal (internal callus) & finally between the cortex of the bone ends (intermediate)



3. Formation of mature bone (ends at 4-6 months)

- The intermediate callus is gradually replaced by lamellar trabeculae laid down along the lines of stress and strain.
- The external and internal calluses are absorbed with restoration of the medullary cavity.

Factors affecting union of fractures

1-Age:

- Fractures of children heal in a much shorter time than adults.
- Osteoporosis in elderly people retards the union of fractures.

2- Type of fracture:

- Oblique & spiral fractures heal rapidly than transverse fractures

3-Position of the fragments:

- If the fragments are impacted, union occurs rapidly.
- Distraction of the fragments markedly delays healing.

4- Vascularity of the fragments:

- Impaired blood supply → ischemia → avascular necrosis → delayed healing
- Examples: a) Fractures of the lower thirds of the tibia and ulna
- b) Intracapsular fracture of the neck of femur (head of femur becomes avascularized)

5- Immobilization of the fragments (vital for proper healing)

Lack of proper immobilization → repeated movement at the fracture site → interfere with the process of union

6- Infection (disastrous to the healing process)

Infection → granulation tissue destruction & decalcification of bone ends → nonunion

Treatment of fractures

I) General management

- 1-Follow the guidelines of trauma life support: ABCD
- 2- Pain: should be immediately relieved by local splinting (at the accident site) and by analgesics
- 3- Blood loss: must be replaced
- 4- Attention to associated injuries
 - e.g., injuries of the urinary bladder in fractures of the pelvis
- 5- Tetanus toxoid & antibiotics for patients with compound fractures

II) Local management (reduction, fixation and rehabilitation)

1. Reduction of the fracture

- Aim: restoration of normal (not always achieved especially when using closed means) or acceptable anatomy
- Time: Early before the part gets swollen
- Methods:

a) Closed reduction

i) Gravity

- The muscle power of the upper limb is not very strong.

- All fractures are associated with some blood loss that might be massive as in fractures of major long bones, the pelvis & the spine
- Bleeding may not be immediately obvious.
- A patient with a fracture of pelvis or shaft of the femur can lose up to 2 liters of blood
- The management of internal haemorrhage and visceral injury takes priority over a limb fracture
- Reduction is not necessary when the displacement is:
 1. trivial (e.g. a fractured metacarpal → neighbouring bones & soft tissue splint the fragments)
 2. Displacement will not leave functional or cosmetic disability (e.g. , most fractures of the clavicle).
- Reduction is urgent in fractures with complicated vascular or nerve injury.
- Accuracy of reduction is desired in any fracture, particularly fractures involving articular surfaces.

-Fractures like surgical neck or shaft of humerus can be reduced relying on the weight of the limb by placing it in a collar and cuff

-A plaster of Paris cast may be applied to increase weight and prevent side-to-side displacement.

ii) Closed manipulation

- Better done under general anaesthesia → painless & allow muscle relaxation → good alignment of fracture fragments.

iii) Traction

- Used for fractures of long bones of LL, e.g. fracture shaft femur.

- Types → Skin or skeletal traction

b) Open (surgical) reduction.

-Indications:

1. Inability to achieve closed reduction due to interposition of soft tissues between the fractured segments.

2. If it is impossible to reduce the fracture because of inability to hold one of the fracture fragments.

3. Intra-articular fractures as closed reduction will be inaccurate

4. Late unreduced fracture

5. When internal fixation is needed

2. Fixation of the fracture

-Aim: keep the two ends of the fracture in position after reduction until solid healing occurs.

- Types:

a) External fixation, methods:

i) Plaster of Paris

- Should include the joints adjacent to the fracture site.

- Advantages: it is cheap, safe & does not need extensive facilities.

- Disadvantages:

- Joint stiffness.
- Muscular atrophy (due to prolonged immobilization)
- Inability to maintain reduction during the immobilization period
- Liability to cause compartment syndrome or Volkmann's ischemic contracture.

ii) Traction.

- Types: skin or skeletal traction

Skin traction

-Method: an adhesive bandage is applied to the skin and then a weight is attached to the bandage.

- Disadvantage: only a small weight (not > 2.25 Kg) can be applied, therefore, it is suitable only for children.

Skeletal traction

-Method: a special pin is passed into the bone and then a weight is applied to the pin.

-Indications: in adults for fractures of the lower limb.

-Immobilization continue until there is evidence of clinical healing

-Angulation, rotation & overlap are not permissible in the lower limb & forearm

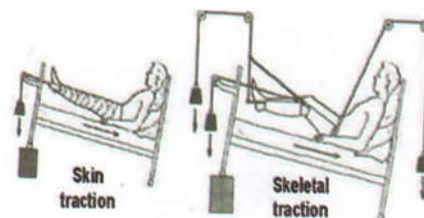
.However, partial side displacement is accepted in a transverse fracture of long bone shafts

-Compartment syndrome:

1. In the first few hours after application of the plaster, edema occurs at the fracture site and as the plaster is inelastic → tissue pressure rises → Venous obstruction → arterial obstruction (if the condition is not treated) → ischemic necrosis of the muscles

2. To avoid this complication, the limb should be padded with wool & the plaster should not be tightly applied.

3. The patient should be kept in hospital for 24 hours to observe peripheral limb circulation & if any sign of ischemia appear → immediately remove the plaster



iii) External skeletal (metal) fixation.

-Indication: useful for compound fractures.

- Method: Special pins are introduced in the bone proximal and distal to the site of the fracture and the pins are connected to a special metal frame

-Advantages: 1.avoids operative internal fixation with the possibility of infection

2. Leaves the wound accessible for inspection.



b) Internal fixation

-Methods:

1. Screws
2. Plate and screws
3. Intramedullary nail
4. Compression screw and plate

-Indications:

i) Difficult fractures

1. Those prone to non-union, especially the femoral neck.
2. Those prone to malunion, e.g. fracture-subluxation of the ankle and wrist (are often unstable) and mid-shaft fractures of forearm.
3. Those prone to be pulled apart by muscle action, e.g. transverse fractures of patella and olecranon.

ii) Pathological fractures (2^{ry} to metastasis or osteoporosis)

iii) Multiple fractures (with 2 major fractures in one limb, fixation of one facilitates closed treatment of the other)

iv) Unstable fractures

v) Nursing difficulties

E.g., elderly patient, Internal fixation allows early ambulation in order to reduce the risks of DVT, bed sores and pneumonia

VI) Associated soft tissue injuries, e.g. vessels or nerves

- Advantages:

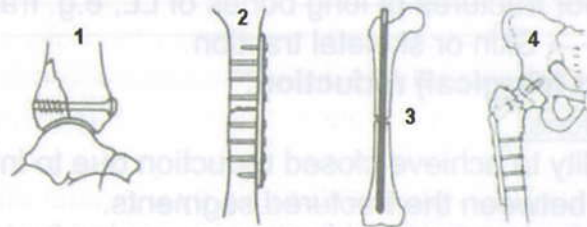
1. Very accurate reduction of the fracture segments.
2. Strict immobilization of the fractured bones.
3. Early mobilization of the patient thus avoiding all the problems of prolonged immobilization particularly in elderly persons.
4. Protection of vascular anastomoses in vascular injury.

3 -Rehabilitation

-Aim: restore function of injured part & that of the patient as a whole.

-All unsplinted joints must be used from the very start and the muscles controlling these joints are kept active by exercises.

-After removal of the splints, residual joint stiffness is treated by graduated exercises.



- Fracture fixation should precede vascular or neural repair, or else the site of repair would be subject to disruption.

- In compound fractures:

1- Nerve injury → mark it for delayed repair

2-blood vessel injury →ligate, repair or graft

Management of compound fractures

- Treatment is urgent surgery→ debridement (excision) of dead tissues followed by external fixation.
- Steps: 1-Done in an operating theatre under general anaesthesia.
- 2-The wound is thoroughly cleansed.
- 3- The deep fascia is widely opened.
- 4- Any foreign bodies, necrotic tissue, necrotic muscles, or necrotic skin edges are removed.
- 5-Any soft tissue injury is dealt with according to the rules
- 6-Bleeding points are tied but a major vessel injury is repaired
- 7-The deep fascia and skin are closed if there is no contamination. (if contamination is suspected or surgery is delayed for more than a few hours, they are best left open)
- 8-Stabilize the fracture externally by a plaster slab or external skeletal fixators (internal fixation is avoided for fear of infection)
- 9-Prophylactic antibiotics and prophylaxis against tetanus

Complications of fractures

1) General Complications

1- Shock

- Neurogenic and hypovolaemic shock may accompany major fractures as those of the spine, pelvis or femur.
- Blood loss with a fractured pelvis may be up to 2-2.5 liters

2- Respiratory complications

- Prolonged recumbency (particularly in elderly) → aspiration pneumonia and pulmonary embolism.
- Incidence reduced with early mobilization of patients following recent techniques of open reduction and internal fixations

3-Deep vein thrombosis

4- Fat embolism

5-Urinary calculi

- Cause: Prolonged immobilization → demineralization of the skeleton → formation of calcium phosphate calculi.

- Prevention: High fluid intake and early mobilization

6- Bed sores

- Cause: secondary to prolonged immobilization especially in elderly
- Prevention: Frequent change of the position, massage, assurance of dry bed sheets and the use of an air-mattress

7- Tetanus (in compound fractures)

-Fat embolism:

May follow multiple or major fractures where fat droplets from bone marrow or adipose tissue enter an artery → distal embolization.

-Timing: usually one day after injury

-C/p:

1. Cerebral embolization makes the patient drowsy, restless & finally, comatose.
 2. PE →dyspnoea and cyanosis.
 3. Petechial rash
- Diagnosis is essentially clinical. However, it may be confirmed by finding fat droplets in sputum & urine
- Treatment: by respiratory support, heparinization and low molecular weight dextran

- It is essential to check the circulation in the distal part of the limb and if there is suspicion of a vascular injury, an angiography may be performed.
- If an arterial injury is diagnosed, open reduction & internal fixation are performed prior to vascular repair.
- Venous injuries are also repaired

II) Early local complications

1. Skin injury

-The fractured bone ends may injure the skin from inside (internal compound fracture), but fortunately the risk of infection is not high

2. Vascular injuries

-Causes:

- i) The fractured bone ends may injure the adjacent vessels
As in: a) Injury of the brachial vessels following a supracondylar fracture of the humerus
b) Injury of the popliteal vessels as a consequence to supracondylar fracture of the femur
- ii) Tense haematoma may damage the artery
- iii) Contusion with 2^{ry} thrombosis

3- Nerve injuries

- Mechanism of injury: excessive traction or compression
- Result: neurapraxia, axonotemesis or neurotemesis (less common)
- Examples: a) circumflex nerve injury in fracture neck humerus b) radial nerve injury in fracture shaft humerus

4- Tendon or muscle injury

5- Infection (serious complication)

- Cause: may complicate compound fractures.
- Effect: leads to delayed healing, non-union or osteomyelitis.

6- Avascular necrosis of bone

- Cause: some fractures damage blood supply to some bone areas
- The commonest example is intracapsular fracture of the neck of the femur → avascular necrosis of femur head → delayed union or non-union of the fracture or osteoarthritis of hip joint (later)

7- Visceral injury

- Example: pelvic fracture → injury to the bladder or Urethra

III) Delayed local complications

1- Malunion

- Definition: union but with some deformity as angulation
- Cause: a) Inadequate reduction b) Failure of immobilization
- Avoided by: Open reduction and internal fixation
- Effect: lead to cosmetic and functional disability

2- Delayed union and nonunion

- Causes:
 - a) Impaired blood supply of one or both fragments (as in intra capsular femoral neck fracture) → poor invasion of the fracture haematoma by cells that have osteogenic potential with decreased ability to produce bone due to low oxygen tension
 - b) Inadequate immobilization → cutting of granulation tissue
 - c) Interposition of soft tissue between the fragments.
 - d) Over-distraction of the fragments due to excessive muscle pull
 - e) Infection which destroys the granulation tissue.
 - d) Pathological fractures, e.g. due to malignant disease



Delayed healing

- Bone ends are decalcified and the fracture line is widened into a gap full of fibrous tissue.
- Clinically: still abnormal movement & tenderness at the site of fracture
- Spontaneous healing is possible with prolonged, uninterrupted immobilization

Nonunion

- Bone ends are sclerosed & the medullary canal is closed by dense Bone
- Spontaneous healing is impossible
- Treatment of delayed union & non-union done by some form of internal fixation and application of autogenous bone graft (from cancellous tissue of the iliac crest)

3- Sudek's atrophy

-Definition: This is a syndrome of osteoporosis, swelling of the soft tissue, vascular stasis, pain & joint stiffness complicating a fracture.

-Etiology: unknown, supposed to be due to:

- a) Reflex vascular stasis due to sympathetic overactivity
- b) Disuse atrophy (patient reluctant to use limb after splint removal)

-Commonest example: following Colles' fracture.

-Treatment: splinting, sympathetic block, analgesics & physiotherapy

4- Myositis ossificans

-Mechanism: a) extensive stripping of the periosteum & ossification of the subperiosteal haematoma

b) or due to heterotopic bone formation in adjacent muscles.

-Commonest examples: after dislocation of elbow, shoulder or hip.

-Treatment: a) Avoid Massage

b) Immobilization (allow haematoma stabilization & new bone resorption)

5- Joint stiffness and osteoarthritis

-Causes: a) common after intra-articular fractures especially in elbow, shoulder & hip

b) After prolonged immobilization

6-Growth disturbance if fracture affects epiphyseal plate in children

7- Osteoporosis: due to prolonged immobilization

Injuries of joints

1- Ligament injuries

	Sprain	Complete tear
Definition	Some fibers injured	All fibers injured
C/P	1. Limited movement 2. Severe pain 3. Tender injury site	Haemarthrosis
Joint stability	Stable	Un stable
Treatment	Joint is firmly bandaged until pain subsides	1. Haemarthrosis is aspirated. 2. Plaster of Paris cast for 6-8 weeks. 3. Later active exercises avoiding tension on the ligament. 4. In young individuals → early surgical repair is favored

- Assessment of joint stability is done under anaesthesia

2- Traumatic effusion

- Source: synovial membrane produces effusion following Joint injury
- Timing: usually several hours after the injury
- The commonest joint affected: knee.
- Treatment:
 - a) Bandage or splint is applied to limit the effusion & relieve pain
 - b) If effusion tense → aspiration

3- Haemarthrosis

- Definition: bleeding inside the joint
- Etiology: severe injury (gross ligament injury or intra-articular fracture)
- C/P: swollen joint with severe pain & limited mobility
- Treatment:
 - a) Aspiration of blood.
 - b) Joint immobilization for a few days by a bandage or a splint.
 - c) Active muscular exercises

4- Internal derangement of a joint

- Definition: various injuries which impair joint movements & stability.
- The commonly affected joint: knee
- Types of injuries:
 - a) Injury of the medial, lateral or cruciate ligaments.
 - b) Injury of the menisci.
 - c) Loose bodies.
 - d) Recurrent dislocation of the patella.

5- Dislocation and subluxation

- Dislocation: joint surfaces are completely displaced and are no longer in contact
- Subluxation: there is partial disruption of the joint and the articular surfaces are still opposed

Upper limb

Fracture Clavicle

Incidence

The commonest fracture in the whole body

Trauma

Indirect → fall on the outstretched hand

Site

Always occurs in the middle third (the weakest part)

Displacement

The outer fragment: displaced downwards & inwards by arm weight

The inner fragment: pulled up by the sternomastoid

- In children, the fracture is often of the greenstick variety.

Complications

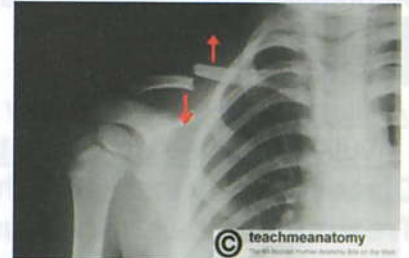
- 1- Malunion (common)
 - Of no functional significance, rarely has cosmetic problem
- 2- Non-union (uncommon)
- 3- Injury of the subclavian vessels and brachial plexus (rare)

Clinical picture

- 1-The shoulder is sagging and deformed.
- 2- Characteristic posture (position of lactating mother)
 - The patient usually supports his elbow with the opposite hand and bends his head to the affected side (to relax the sternomastoid)

Treatment

- 1- A broad arm sling for **3 weeks** and analgesics.
- 2- Internal fixation (If there is associated vascular or nerve injury)



Shoulder dislocation

Anterior dislocation

Incidence

The commoner type (Posterior dislocation is rare)

Trauma

Forced extension and external rotation of the abducted arm

- The shoulder joint has sacrificed stability for mobility.
- Shoulder instability is due to:
 - a) Shallow glenoid cavity with a relatively large humeral head.
 - b) Lax capsule
 - c) Lack of support by strong ligaments & muscles

Displacement

- The head of humerus: dislocates forward → subcoracoid position → the humerus is locked in place by muscle spasm
- The capsule of the shoulder: ruptures its anterior aspect
- The labrum glenoidal: avulses from the glenoid.
- The humerus passes over the anterior margin of the glenoid → compression fracture of posterior part of articular surface of the head

Complications

- 1- Axillary nerve injury: mostly neurapraxia → spontaneous recovery
- 2- Avulsion of the supraspinatus tendon
 - **Discovered by** inability of the patient to initiate abduction
 - **Treatment**: repair of the musculotendinous cuff.
- 3- Associated fracture of greater tuberosity or humeral neck.
- 4- Recurrence (a common problem)

Clinical picture

Inspection

- 1- The outer aspect of the shoulder is flattened.
- 2- The arm appears to take origin from a point under the junction of the middle and outer thirds of the clavicle.
- 3- The patient supports the elbow of injured arm by the other hand in slight abduction of the arm
- 4- The distance between the point of the elbow and the axillary skin is reduced and the axillary concavity is obliterated.

Movement

The shoulder cannot be moved

Investigation

X-rays in anteroposterior views → diagnostic

Treatment

Reduction under general anaesthesia with muscle relaxation

Kocher's technique

1. Downward traction (to disengage the head)
 2. External rotation (to overcome subscapularis muscle spasm)
 3. Adduction (bringing the elbow across the chest)
 4. Internal rotation (carrying the hand to the opposite shoulder)
- Reduction is confirmed by putting the shoulder through a full range of movement under anaesthesia



Recurrent shoulder dislocation

- **Etiology**: due to detachment of labrum glenoidal & the anterior capsule from the anterior margin of the glenoid → the humeral head slips easily through the torn capsule
- **Mechanism**: with shoulder abduction & external rotation (occurs with increasing ease & frequency) during minor event as combing hair
- Reduction is equally easy and usually patient can reduce the dislocation himself
- **Treatment**: Surgically by repair of the anterior capsule & subscapularis muscle if it were also torn.

Immobilization

- For **3 weeks** in adduction & internal rotation of the shoulder by a sling and bandaged
- Allow healing of torn capsule and minimize recurrence tendency

Rehabilitation

At the end of 3 weeks, the sling is removed and progressive mobilization of the shoulder is done for **3 more weeks** (avoid only external rotation in abduction → position of shoulder dislocates)

Fractures of the humerus

Fractures of the neck of humerus

Mechanism of injury

- Indirect: by a fall on the outstretched hand
- Direct: fall on the point of the shoulder.

Classification

- 1- Stable
- 2- Unstable (caused by comminution)

Clinical picture

Type of patient

- 1- Common in the elderly
(Usually associated with osteoporosis & only need minor trauma)
- 2- In younger adults
(Require a considerable force & fracture dislocation may occur)

Symptoms

Pain in the shoulder and inability to move the joint

Complications

1-Shoulder stiffness

-Common especially in the elderly who may never regain a full range of movements.

-Treatment → early passive followed by active mobilization.

2- Axillary nerve injury.

3- Axillary artery and brachial plexus injury.

4- Malunion (common but with excellent functions).

5- Avascular necrosis of the humeral head.

6- Nonunion (not common).

7-Associated shoulder dislocation (dislocation should be treated first)

Fractures that occur due to fall on the outstretched hand

1. Clavicle fracture
2. Supracondylar (extension type)
3. Posterior elbow dislocation
4. Colles' fracture
5. Scaphoid fracture



- Diagnosis is made radiologically
- Limited movement may be possible in minor impacted fractures

Treatment

1- In stable fractures (particularly in the elderly)

- By an arm sling and as pain diminishes within days → gently move the shoulder (to avoid stiffness)

2- In unstable comminuted fractures (in young patients)

a) Mild or no displacement → closed reduction under anaesthesia

b) Marked displacement → an open reduction and internal fixation



Fractures of the shaft of the humerus

Trauma

1- Indirect :

a) Arm twisting → spiral fracture with or without a butterfly fragment.

b) Fall onto the outstretched hand.

2- Direct:

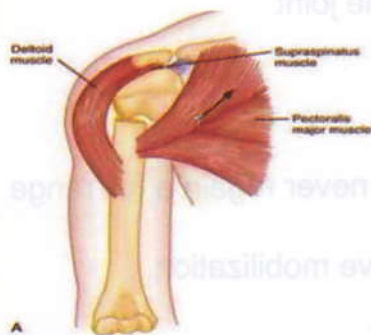
Blow over the shaft → transverse fracture (+/- butterfly fragment)

Butterfly Fragment



Displacement

	Upper 1/3 (just below the neck)	Middle 1/3	Lower 1/3
Proximal fragment	abducted by the supraspinatus	adducted and pulled inwards by the pectoralis major	abducted by the deltoid
Distal fragment	adducted by the pectoralis major	abducted by the deltoid	adducted and pulled upwards by the coracobrachialis



Complications

1- Delayed union and nonunion.

2- Joint stiffness

3- Radial nerve injury

- Mostly neurapraxia → full recovery in most cases.

- Motor: wrist drop and finger drop

- Sensory: localized to snuff box



I-Closed methods

A) Reduction & fixation using → U-shaped plaster slab (for **8 weeks**)

- Reduction obtained by gravity acting on the arm and exerting steady traction → correct any angulation & overriding that may occur
- The bandage runs from the top of acromion, down the lateral side of the arm, under the elbow and up the medial side of the arm to axilla.
- For further stability → the arm bandaged to the chest wall.

B) Alternatively → a hanging cast

(A complete cast extending from the axilla to the wrist)

- Advantages: the action of gravity is more because of its heaviness
- Disadvantages: the elbow cannot be mobilized during treatment

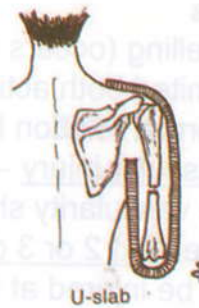
II- Open reduction and internal fixation (rarely needed)

- Indication: when closed methods not obtain a satisfactory reduction

III-Treatment of complications

Radial nerve injury

Avoid development of fixed flexion of the wrist and fingers by a combination of a removal cock-up splint and passive mobilization



Supracondylar fractures

Types

	Extension type (The commonest)	Flexion type (Rare)
Trauma	Fall on the outstretched hand with the elbow slightly flexed	Fall on a flexed elbow.
Displacement	-The distal fragment is displaced and angulated posteriorly in relation to the proximal one.	The distal fragment is displaced anteriorly by biceps & brachialis

- The fracture line runs transversely through the distal metaphysis of the humerus.
- The fracture is greenstick in **50%** and complete in **50%** of cases.



Clinical picture

Type of patient

More frequent in children

Symptoms

- 1- Pain
- 2- Swelling at the elbow region.
- 3- The child is not able to move the elbow.

Signs

- 1-Swelling (occurs rapidly within **3 to 4 hours** →obscure other signs)
- 2- Limited both active and passive movements
- 3- Normal relation between 3 bony points at elbow
- 4- Vascular injury →**the brachial artery**
 - The vascularity should be examined initially and then at intervals for the next 2 or 3 days
 - May be injured at time of trauma by the anterior aspect of the distal end of proximal fragment → radial pulse is not felt from the start
 - More commonly the radial pulse is palpable at the time of the injury but is lost by swelling and flexion of the elbow following reduction.

Complications

1-Nerve injury

- Injury of the median, ulnar and radial nerves may occur.
- It is usually of the neurapraxia type → conservative treatment

2- Brachial artery injury

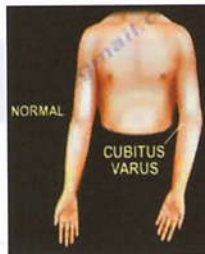
- **Effect:** a) Gangrene b) Volkmann's ischemic contracture
- c) Acute compartment syndrome (may occur with adequate distal pulse) → treated by Fasciotomy

3- Elbow stiffness

4- Malunion → producing cubitus varus

- Cause:** improper reduction

5- Myositis ossificans



Treatment

I-Closed reduction and fixation (the standard treatment)

- By a posterior slab for **3-4 weeks** followed by gentle mobilization
- Done urgently under anaesthesia before gross swelling makes palpation of the normal anatomy impossible.
- The usual extension type is fixed in flexion.
- Then above elbow packed posterior slab is applied
- The reduction is checked radiographically, if satisfactory → add collar and cuff

II-Closed reduction and percutaneous pinning

III- Open reduction and internal fixation (using wires)

- Indications: unsatisfactory reduction by closed methods

Rehabilitation

Active elbow movement and physiotherapy

- If no significant swelling → posterior prominence of the point of the elbow is obvious & can palpate the medial and lateral epicondyles and the point of the olecranon → normally related to each other (to differentiate from posterior elbow dislocation)

-Throughout the gradual reduction process the radial pulse is felt and if it is obliterated by flexion → gradual elbow extension until the pulse returns

- Hospitalization for 48 hours for observation of hand circulation

*If at any time the circulation is felt to be impaired → remove the slab and elbow is extended,

If there is no improvement → explore the brachial artery

Elbow dislocation

Trauma and displacement

Posterior dislocation (the usual type)

- Trauma:** fall on the outstretched hand with the elbow slightly flexed.
- The coronoid process of the ulna is displaced posteriorly behind the distal end of the humerus.
- Spasm of the triceps muscle then locks the elbow in a position

Clinical picture

Symptoms

Inability to move the elbow from a position of slight flexion

Signs

- The point of the olecranon felt posterior to the humeral condyles
- The triangle formed by the tip of the olecranon and the condyles is no longer equilateral (to differentiate from supracondylar fracture)

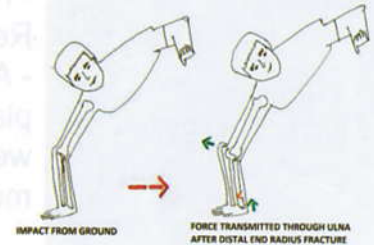
Complications

- 1- Associated fracture of the coronoid process
- 2- Irreducible dislocations (rare & treated by open reduction)
- 3- Median or ulnar nerve damage.
- 4- Brachial artery injury (unusual)
- 5- Myositis ossificans.

Treatment

Closed reduction and fixation

- 1- Under general anaesthesia → traction in the long axis of the slightly flexed forearm.
- 2-The reduction is stable because of the elbow bony configuration.
- 3- Immobilization for 3 weeks in an above the elbow posterior slab (To allow healing of the capsule and ligaments)
- 4- Followed by gradual mobilization



Fractures of the forearm bones

Fracture of olecranon process

Trauma

Fall onto the point of the elbow

Displacement

Separation of fracture ends depends on integrity of the triceps expansion:

- a) If the triceps expansion is intact → no separation
- b) If the expansion is torn → the proximal fragment will be pulled up producing a wide gap.



	Intact triceps expansion	Torn triceps expansion
Clinical picture	Bruising, swelling and tenderness	
Active elbow extension	possible	Lost
Palpable gap	Not detected	Detected
Treatment	- No need for Reduction - An above elbow plaster cast for 6 weeks is followed by mobilization	By open reduction and internal fixation followed by early movement of the elbow 

There is great similarity between fractures of olecranon and those of the patella

Fractures of the shafts of radius and ulna

Fracture of both bones

Trauma

A) Direct: blow to the forearm (break the bones at the same level)

B) Indirect: twisting of the forearm → oblique fractures situated at different levels in the two bones.



Displacement

• Besides overlap, angulation and side displacement, rotation may occur (due to unbalanced pull of the supinator and pronator muscles attached to the radius)

Site

• The upper, middle or lower thirds of the forearm.

Treatment

A) Undisplaced fractures

By a full arm plaster cast with 90° elbow flexion and neutral rotation, for approximately **6-9 weeks**.

B) Displaced fractures.

1- In adults → open reduction and compression plating is done (If reduction is not perfect → loss of pronation and supination)

2- In children → closed reduction and plaster fixation

(Residual angulation of about 5° accepted → will be corrected by remodeling)

Fracture of one bone without angulation

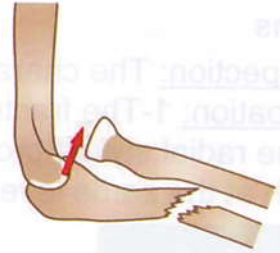
-Trauma: by direct trauma.

-Treatment: immobilization in an above elbow plaster

a) In an adult → for **6 weeks**
b) In a child → for **3 weeks**.

Complications

- 1- Malunion
- 2- Nerve injury (uncommon)
- 3- Acute compartment syndrome (treated by Fasciotomy)
- 4- Synostosis (cross-union) between radius and ulna



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Monteggia fracture dislocation

Definition

Fracture of the ulna and dislocation of the superior radioulnar joint

Types

1-Extension type 60%

It consists of anterior angulation (apex anterior) of the ulna and anterior dislocation of the radial head.

2- Flexion and lateral types (less common)

Treatment

- 1- ORIF of the ulna and closed -reduction of the radial head
- 2- If closed reduction fails → open reduction of the radial head and repair of the annular ligament
- 3- Followed by the application of a full-arm plaster cast for **6 weeks**

Complications:

Posterior interosseous nerve injury
- The fracture may be missed in children, the ulna may not fracture. It may just bend enough to allow the radial head to dislocate.

Colles' fracture

Definition

This is a fracture of the distal end of the radius with fracture line within one inch of the lower end of the radius in its cancellous part.

Trauma

Fall on the palm of the outstretched hand

Displacement

The distal fragment shows the following displacement

- 1- Dorsal displacement and tilt.
- 2- Radial displacement.
- 3- Upward displacement and impaction.

Clinical picture

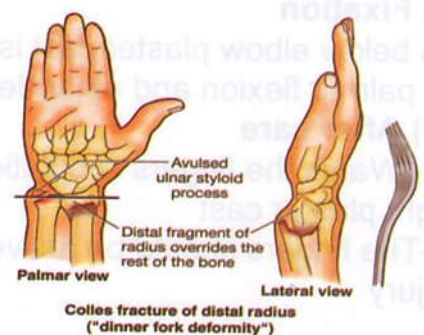
Type of patient:

- Uncommon in < 50 years old
- Later, it becomes increasingly common particularly in women (due to postmenopausal osteoporosis)

Symptoms

Pain and swelling

The fracture is often comminuted, and commonly associated with injury to the Inferior radioulnar joint & ulnar styloid process, which is avulsed



Signs

- Inspection: The characteristic '**dinner fork**' deformity
- Palpation: 1-The fracture site is tender.
- 2-The radial styloid process is no longer distal to the ulnar styloid (Both at the same level → reflecting shortening of radius)

Investigations

- X-rays is diagnostic

Complications

1-Malunion

- There is often some residual deformity because:
 - a) Re-displacement inside the cast especially in comminuted posterior cortex
 - b) A Colles' plaster does not control the supination element of the deformity

- In most cases physiotherapy is all that is needed.

2- Stiffness of the wrist, shoulder and fingers.

3- Carpal tunnel syndrome (late complication)

- Diagnosed by → nerve conduction studies.
- Treated by → surgical division of the flexor retinaculum

4- Rupture of the extensor pollicis longus tendon

5-Sudeck's atrophy

Treatment

I) Reduction

- 1-Done under anaesthesia
- 2-An assistant grasps the upper arm while the surgeon grasps the injured hand as if '**shaking hands**'.
- 3-The manipulation is carried out by **3 grips**
 - a) Traction along the long axis of the limb → disimpact the fragments
 - b) Pushing the distal fragment towards the ulna → correct the radial displacement and tilt.
 - c) Pressing the distal segment anteriorly with palmar flexion and pronation → correct the posterior displacement and tilt

II) Fixation

A below elbow plaster cast is applied for **6 weeks** holding the wrist in palmar flexion and ulnar deviation

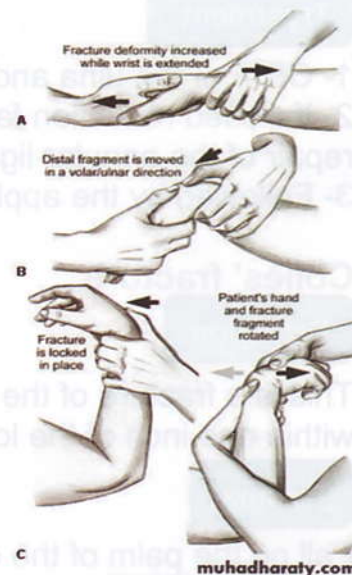
III) After care

- 1- Watch the fingers circulation in the initial 24 hours for fear of a tight plaster cast
- 2-The fingers should be actively mobilized immediately after the injury



Dinner fork deformity

- From the side → dorsal aspect of the wrist is prominent
- From the Dorsum → lateral aspect of the wrist is prominent & hand is radially deviated



- The patient should be instructed to elevate the arm above the head several times daily → prevent stiffness → improve functional outcome (more important than the treatment of the fracture itself)

Fractures of the hand

Fracture of the scaphoid

Trauma

Fall on the outstretched hand → fracture usually at the bone waist

Clinical picture

Type of patient: typically in young adults

Symptoms

- 1-Pain in the wrist region
- 2-The joint function may not be markedly impaired.

Signs

- 1-Tenderness over the scaphoid in the anatomical snuff box
- 2- Little swelling
- 3- No bruising

Investigations

X-ray (AP, lateral and oblique views)

-May not be informative immediately after the injury because no displacement occurs at the fracture site.

Complications

- 1- Non-union (treated by bone grafting +/- internal fixation)
- 2- Avascular necrosis of the proximal fragment (because blood supply to the proximal part of the bone enters through distal end)

Treatment

- 1-If suspected → scaphoid plaster should be applied for **2 weeks** then the wrist is re-x-rayed (by this time, bone resorption occurred at the fracture site → radiologically evident)
- 2-The wrist is fixed in a below elbow cast, including the proximal phalanx of the thumb for **8 weeks**
(Bony union occur in 90% of uncomplicated cases)

Fractures of the phalanges

-Fracture lines: usually transverse

-Displacement: forward angulation by the tension of the lumbrical and interosseous muscles.

-Treatment: correct the angulation & the finger is immobilized in semi flexion over a finger wire splint incorporated in a forearm plaster cast

Mallet finger

-Definition: This is avulsion of the extensor expansion from the base of the terminal phalanx resulting in flexion deformity of that phalanx.

-C/P: The patient cannot actively extend the DIP joint

-Treatment: The finger is immobilized with the PIP joint in flexion (so as to relax the lateral slip of the extensor expansion) and the DIP joint hyperextended (so as to approximate the end of the tendon to the raw area) for **6 weeks**.

Physical signs are often slight and suggest a sprained wrist rather than a fracture → often missed and wrongly diagnosed



Fractures and dislocations of lower limb

Fractures of the pelvis

Incidence

- 1- Constitute about **1.5%** of all fractures.
- 2- Mortality rates vary from **5% to 50%** depending on:
 - a) Severity of disruption
 - b) Associated injuries.
- 3- Most fractures occur in young adults involved in high energy trauma as road traffic accidents.
- 4- Often associated with other serious injuries

Classification

According to

A) Type of trauma

- 1- Forces directed from the front or the back → anteroposterior compression or open book injuries
- 2- Laterally directed force → lateral compression
- 3- Vertically directed force → vertical shear (unstable injuries)
- 4- Combined mechanical injuries.

B) Stability of the fracture

I- Stable fractures

-**Definition:** fractures stable in rotational & vertical directions do not usually cause complications

-Examples:

- 1- Fractures outside the pelvic ring as
 - a) Avulsion injuries of the ASIS or AILS or ischial tuberosity
 - b) Fractures of the sacrum
 - c) Fractures of the coccyx
- 2- Single fractures of the pelvic ring

II- Unstable fractures

1- Open book fracture (Hinge subluxation)

-It is rotationally unstable but vertically stable

-**Cause:** trauma that is either → a) direct anteroposterior

- b) Lateral compression from a crushing injury
- c) Indirect lateral compression through the lower limb

-**Effect:** Disruption of the symphysis pubis with wide separation in addition to disruption of one sacroiliac joint with little separation

2- Vertical shear fracture (**has highest mortality and morbidity**)

- Both rotationally and vertically unstable

-**Consists of:** a) **Posteriorly** → fracture of ilium or sacrum or sacroiliac joint disruption

b) **Anteriorly** → fracture of either rami or symphysis pubis disruption

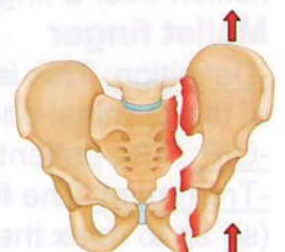
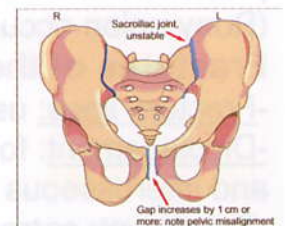
- The anterior & posterior trunk muscles pull the mobile hemi-pelvis upwards

- It also opens up like a book by the weight of the leg.

-The pelvis is a rigid osseo-ligamentous ring with considerable stability.

-It consists of the 2 hip bones & sacrum linked by:

- a) **Posteriorly:** the very strong sacroiliac, sacrotuberous & sacrospinous ligaments
- b) **Anteriorly:** the symphysis pubis & related ligaments



Examination

A) General

Shock

B) Local

- Inspection:

- 1- Massive flank or buttock contusions and swelling with hemorrhage
- 2- Lower limb deformity (shortening, external rotation without long bone fractures)

- Palpation: (always examine the patient's back when log rolled)

- 1- Manual stress examination → flexible pelvis
- 2- Palpable displacement through posterior ring

- Neurologic examination:

- Sciatic nerve, L5 and S1 roots especially with sacral fractures.

- Visceral injuries

- 1- GIT abdominal and rectal examination.
- 2- Genitourinary → scrotal hematoma, urethral and bladder injuries

Investigations

- 1- Plain X rays (AP, inlet and outlet views)
- 2- CT → define the fracture lines especially posterior involvement of sacrum & pelvis

Complications

- 1- **Shock** (Most importance)

- Injury of pelvic vessels → severe internal haemorrhage

- 2- **Renal failure** due to hemorrhagic shock

- 3- **Injury of pelvic structures** as injury of:

- | | |
|----------------------|--------------------|
| a) Posterior urethra | b) Urinary bladder |
| c) Rectum | d) anal canal |

- 4- **Injury to pelvic nerves**

- 5- **Complications of prolonged recumbency** as

- | | |
|--------------|-----------------------|
| a) DVT | b) Pulmonary embolism |
| c) Pneumonia | d) Decubitus ulcers. |

- 6- **Secondary osteoarthritis**

- Commonly after disruption of sacroiliac joints & acetabular fractures

Treatment

A) General principles

- 1- Patient primary assessment (ABCDE) & full trauma evaluation
- 2- Correction of the hypovolaemic shock is vital. Remember that
- 3- Associated injuries treatment take priority over the bony injuries.

B) Treatment of fracture

Stable fractures

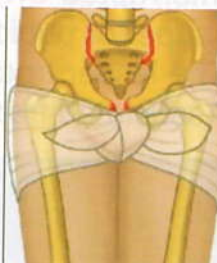
- 1- Rest in bed, analgesics and physiotherapy
- 2- After few days, the patient starts moving out of bed.

- Hemorrhage is the main cause of death in pelvic injuries.

- Sources of bleeding include:
a) The posterior sacral plexus of veins

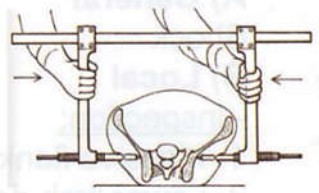
b) Osseous & visceral bleeding

- Fracture of the pelvis may lead to **2-2.5 liters** of blood loss



Unstable fractures

- 1- Initial stabilization should be performed either by simple pelvic binders or external pelvic fixators
- 2- If there is still vascular instability → do angiography & embolization
- 3- If this is unsuccessful → exploration, surgical homeostasis and packing of the pelvis
- 4- Open reduction and internal fixation (the final management)



Hip dislocation

Posterior dislocation

Trauma

- When **hip is flexed and adducted** (In this position, the femur head is covered posteriorly by the capsule rather than bone)
- A force applied in the long axis of femoral shaft → head dislocates backwards over the posterior lip of the acetabulum
- Caused by two common accidents:
 - 1- A weight falling on the back of a person in a stooping position.
 - 2- In car accidents, when the knee hits the dashboard "**dash-board dislocation**".

Clinical picture

Symptoms: Severe pain and swelling

Signs

- Inspection → Deformity:

- a) The hip is **flexed, adducted and internally rotated**
- b) **Shortening** of the affected limb.

- Palpation: 1- greater trochanter is raised

2- Femoral head is palpable in the gluteal region (empty femoral Δ)

- Movement: Hip movements are painful and limited.

Investigations

X-ray shows:

- 1- The femoral head lies outside the acetabulum.
- 2- Interrupted **Shenton's line** (curve formed by the lower margin of the superior pubic ramus and the lower border of femoral neck)
- 3- The lesser trochanter is less apparent (due to internal rotation)
- 4- Associated fractures as chip fracture of acetabular posterior wall



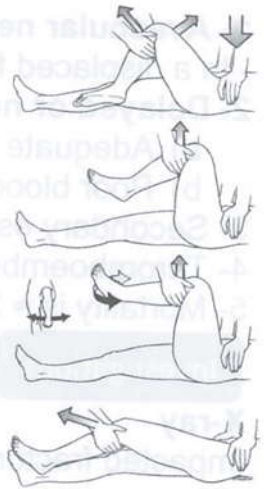
- The hip joint is stable (unlike shoulder joint) because of:
 - a) The depth of the acetabular cavity
 - b) Strong support by its ligaments and muscles.
- Therefore less frequent to be injured
- Types of dislocation:
 - a) Posterior (the commonest)
 - b) Anterior
 - c) Central.
- The hip commonly displaces posteriorly while the shoulder commonly displaces anteriorly

Complications

- 1- Sciatic nerve injury
- 2- Femoral head avascular necrosis (especially in delayed reduction)
- 3- Associated fractures:
 - a) Posterior acetabular rim fracture (the commonest)
 - b) Of the patella and femoral shaft.
- 4- Secondary osteoarthritis

Treatment

- 1- Closed reduction (done under general anaesthesia)
- 2- The patient is placed supine and an assistant steadies the pelvis
- 3- The hip and the knee are flexed → bringing the head of the bone posterior to the acetabulum.
4. The femur is then adducted and pulled vertically upwards → draw the head forwards into its socket.
5. The hip is then extended and the reduction is maintained by skin traction for **3 to 6 weeks**



Fractures of the femur neck

Intracapsular fractures of femoral neck

Site

Portion of the neck of femur which lies within the hip capsule

Classification

A) According to fracture site:

- 1- Subcapital → at the junction of the head and neck
- 2- Transcervical → somewhere in the neck.

B) **According to displacement:** Either impacted or displaced

Trauma

Most commonly in the elderly (because of senile osteoporosis) after trivial trauma as a fall when the foot catches the edge of a carpet

Clinical picture

A) Displaced fractures

- 1- The hip is adducted & externally rotated (by weight of the limb)
- 2- Shortening of the limb.
- 3- The patient is unable to lift the leg off the examination couch.

B) Impacted fractures

No abnormal physical signs other than tenderness over the fracture site → so easily missed clinically

Central dislocation

-Cause: a blow on the greater trochanter → Drives the head of the femur through the floor of the acetabulum → comminuted fracture of the acetabulum (may be accompanied by other pelvic fractures)



Complications

1- Avascular necrosis

- In a displaced fracture due to damage of femoral neck blood supply

2- Delayed or non-union, may occur due to

- Adequate immobilization is difficult (even by internal fixation)
- Poor blood supply to the proximal fragment.

3- Secondary osteoarthritis.

4- Thromboembolism in **25%** of patients.

5- Mortality is $\approx$ **20%** during 1st **3 months** after fracture in elderly

Investigations

X-ray

Impacted fractures with minimal displacement may be missed

Treatment

Operative because:

- The proximal fragment can neither be properly manipulated nor immobilized by conservative means
- Internal fixation helps early mobilization of these elderly patients who are prone to the complications of prolonged recumbency

I-For impacted fractures

- No reduction is required
- Internal fixation with 2 cannulated screws.

II-For displaced fractures

1- Patient < 65 $\rightarrow$ Closed reduction, if failed $\rightarrow$ ORIF fixation as

2- Patient > 65 $\rightarrow$ **Hemiarthroplasty** (treatment of choice)

- The head & neck of the femur are replaced with a metal prosthesis
 - Used because chance of non-union or of avascular necrosis is high

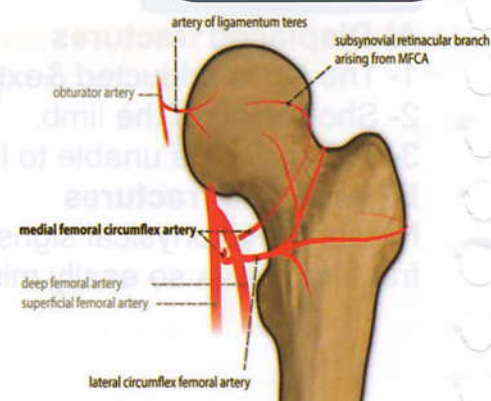
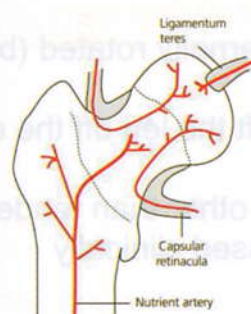
- Postoperative

Early ambulation to all cases once the general condition allows

The blood supply of the head of the femur

From 3 sources

- An extracapsular arterial ring
 - Site: at the base of the neck
 - Formed from: Branches of the medial & lateral circumflex femoral arteries
 - Gives rise to ascending cervical branches (on the surface of the neck, known as the **retinacular vessels**)
- Arteries of the ligamentum teres
 (not always patent in adults, and if are, only supply a small area of bone)
- Terminal branches of the ascending nutrient artery



Extracapsular fractures of femoral neck

Site

Fractures extending from the base of the neck of the femur to **8 cm** below the lesser trochanter

Types

- 1- Trochanteric fractures: down to the level of lesser trochanter
- 2- Subtrochanteric fracture: from the lesser trochanter to 8 cm below

Trauma

-In young person:

Trochanteric & subtrochanteric usually result from major trauma.

-In the elderly:

A fall on the side over greater trochanter → trochanteric fracture

Clinical picture

Symptoms: Inability of the patient to raise his leg.

Signs:

-Inspection → External rotation and shortening of the limb.

-Palpation → Tenderness over the fracture site.

Complications

- 1- Thromboembolism
- 2- Malunion leading to shortening and external rotation.

Treatment

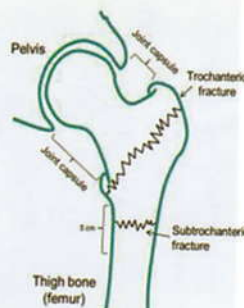
A) Trochanteric fractures

- In the elderly (most frequent)

The best treatment is to do internal fixation by a dynamic hip screw

-In young persons → immobilization and traction.

B) Subtrochanteric fractures → internal fixation by plate & screws



Extracapsular fractures differ from intracapsular fractures in:

- 1- The blood supply of the 2 fragments is good → no avascular necrosis & no nonunion
- 2- The proximal fragment can be controlled conservatively → so ORIF is not mandatory

Fractures of the shaft of femur

Incidence

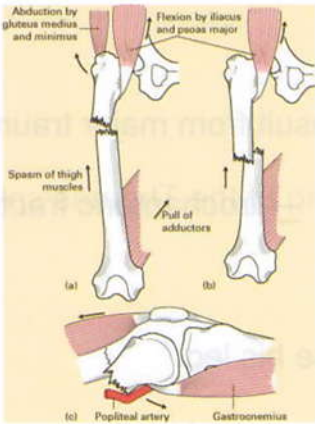
Occur in children and adults

Trauma

Result from severe violence (associated injuries are common)



Displacement

	Upper third	Lower two thirds
Proximal fragment	- <u>Flexed</u> by: iliopsoas - <u>Abducted</u> by: the gluteal muscles - <u>Everted</u> by: the external rotators	Anteriorly angulated by <u>quadriceps</u>
Distal fragment	- <u>Adducted</u> by: the adductor muscles - <u>Pulled up</u> by: the hamstrings - <u>Everted</u> by: weight of the limb. 	Posteriorly angulated by <u>Hamstring</u>

Investigations

X-ray

Performed to the pelvis and knee to rule out associated injuries

Complications

1- Vascular injury

Lower third displaced fractures → injure femoral or popliteal vessels

2- Nerve injury

The outer bar of a Thomas splint may injure common peroneal nerve

3- Non-union (uncommon)

4- Malunion

5- Knee stiffness (serious complication)

Especially after treatment by closed reduction and traction



The strong muscles of the thigh cause overriding of the fragments → consequent shortening.

Treatment

I-General principles

- 1- Proper alignment is important to avoid knee joint osteoarthritis
- 2- Shortening **> 2 cm** should not be allowed

II- Conservative treatment

- Reduce the fracture then keep it immobilized by applying traction to the distal segment and counter traction to the proximal fragment

- There are two methods:

1) Sliding traction

- **Distal fragment traction** by weights and pulleys attached to the limb either by:

a- skin strapping in children

b- skeletal traction in adults

By inserting special pins in the upper end of the tibia

- **Counter traction** depends upon the weight of the body acting by elevation of the foot of the bed.

- In children < 2 years, the same principle applied using **gallows' splints** (strapping of the legs and slinging them to a cross bar, so the pelvis is lifted from the mattress)

2) Fixed traction using a Thomas splint

- **Distal fragment traction** by attaching to it cords that are tied to the foot of the splint

- **Counter-traction** by pressure of the ring of the Thomas splint against the ischial tuberosity and the soft tissues.

- Disadvantages of conservative treatment

1- Prolonged immobilization with all its side effects especially in elderly

2- Liability to stiffness of the knee.

3- If there is soft tissues interposition between the fragments → impossible to perform reduction

4- Ideal traction is difficult (excess traction → distraction)

5- The Common peroneal popliteal nerve injury by compression of the side bar of a Thomas splint.

III- Operative treatment (ORIF)

- This avoids the disadvantages of conservative treatment.

- Indications:

1- Can't perform closed reduction due to soft tissues interposition

2- Associated vascular injury (to avoid disruption of vascular repair)

3- Double-level fractures.

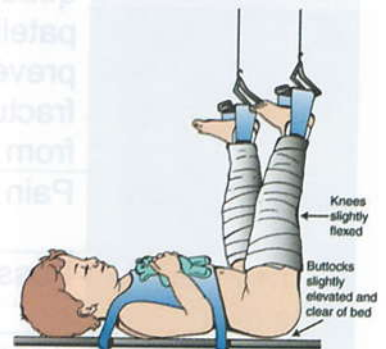
- Methods of ORIF

1- Fractures of the proximal shaft → fixed by plate and screws

2- Fractures of the middle two quarters → by an intramedullary nail

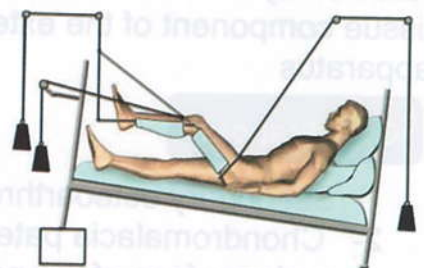
- The tendency for backward angulation of 2 fragments of the femur is controlled by slings under the thigh.

- If the applied weight is not adjusted accurately → distraction of 2 fragments → delayed union



Screws

Plate



Fractures of the patella

Trauma

1- Indirect injury

- Forced knee flexion when the quadriceps muscle is contracting → transverse fracture

2- Direct injury

- Trauma to anterior aspect of flexed knee → comminuted fractures

Types

	1- Un-displaced transverse fractures	2- Displaced transverse fractures
Trauma	Mild	Severe
Bony fragments	-Not displaced - The pre-patellar expansion of the quadriceps tendon & patellar retinacula prevent the two fracture fragments from displacement	- Displaced - The patellar retinacula is destroyed - The quadriceps muscle pulls the proximal patellar segment upwards
Clinical picture	Pain & tenderness	Gap is palpable in the patella
Active knee extension	Possible but painful	Impossible
Treatment	Knee immobilization in plaster for 6 weeks combined with quadriceps exercises.	- ORIF using wires - Early physiotherapy after the operation prevents knee stiffness

3-Comminuted fractures

- Accurate reduction is impossible.
-The best treatment is patellectomy followed by reconstruction of the soft tissue component of the extensor apparatus

Complications

- 1- Secondary osteoarthritis
- 2- Chondromalacia patellae → cartilage on undersurface of the patella deteriorates and softens



Supracondylar fracture of femur

-The gastrocnemius muscle flexes the distal fragment → posterior angulation at the fracture line → popliteal artery injury (Very difficult to treat conservatively)
- The best results are obtained by internal fixation of the fragments by plate & screws followed by early knee mobilization
- Supracondylar fractures of:
a) The femur → injure the popliteal artery
b) The humerus → injure the brachial artery



Injuries of the knee joint

Meniscus tears

Trauma

- It commonly affects foot-ball players when weight is being taken on the flexed knee and there is a twisting strain
- The force grinds the meniscus between the femur & tibia → splits

Types of tear

- 1- Longitudinal (**bucket-handle tear**)
- 2- Transverse (**parrot-beak tear**)

Clinical picture

Symptoms

- 1- A typical history of injury while playing a game → falls, feeling pain in the knee & cannot resume the game but can walk home with a limp.
- 2- Complains of giving way, locking of the joint or recurrent effusion

Signs

- 1- Knee effusion.
- 2- Limitation of complete extension when the knee is locked
- 3- Localized tenderness to the joint line on the medial side.
- 4- Positive **McMurray's sign**
(A click elicited when the knee is rotated in certain degree of flexion)
- 5- Atrophy of the quadriceps muscle (in long-standing cases)

Investigations

- 1- **MRI** (the most reliable)
- 2- **Arthroscopy** → has the advantage that it is diagnostic and therapeutic
- 3- **Plain X-rays** are normal

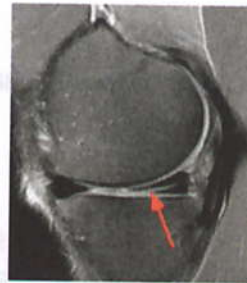
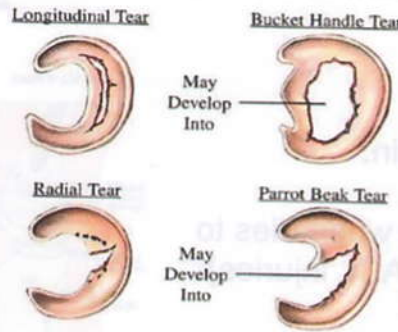
Treatment

A) Conservative treatment

- Indication: if the knee does not lock.
- Method: by rest, ice, compression, elevation & NSAID analgesics.
- The joint is held straight in a posterior slab for **3-4 weeks**
- Quadriceps exercises are encouraged

B) Operative treatment

- Indications: 1- The joint cannot be unlocked.
- 2- Recurrent symptoms as pain, swelling (effusion) or locking.
- Operation:
 - 1- Meniscectomy (by open or arthroscopic technique)
 - 2- The damaged part of meniscus & loose fragments are removed
 - 3- Postoperative quadriceps strengthening exercises



Functions of knee menisci

- 1- Increase knee stability
- 2- Control complex rolling & gliding actions of the joint
- 3- Distribute load during movement

Pathological anatomy

- 1- Meniscus injuries are more frequent on medial than on lateral side
Because:
 - 1- Medial knee compartment carries about 90% of the load during weight bearing
 - 2- Medial meniscus is much less mobile than the lateral (because it is attached to the capsule)

- A meniscus is avascular and a tear does not heal unless it is peripheral (outer third) which is vascularized from the capsule

Knee ligament injuries

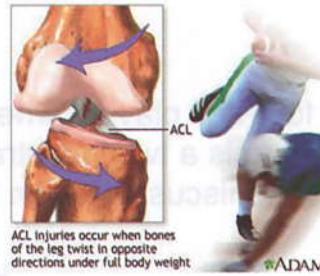
Trauma

1-The ACL (more common)

- Torn by a sudden twisting motion.

2-The PCL (less frequently)

- When ligament pulled or stretched too far as by a blow to the front of the knee



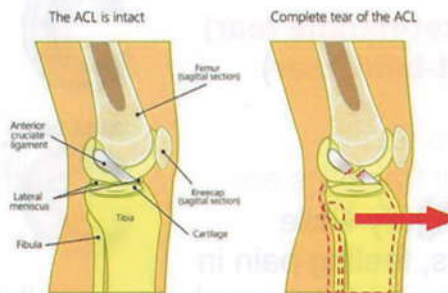
Clinical picture

Symptoms

- 1- May not cause pain.
- 2- A popping sound.
- 3- The leg collapses when tries to stand on it (more in ACL injuries)

Signs

- 1- Joint swelling and tenderness.
- 2- Abnormal joint mobility:
 - a) With ACL tears → tibia excessively glides anterior to the femur
 - b) With PCL tear → tibia excessively glides posterior to the femur



Investigations

- 1- Plain X-ray (to exclude fractures)
- 2- MRI
- 3- Arthroscopy

Treatment

A) Conservative treatment

Similar to that for meniscus injury (may resolve symptoms)

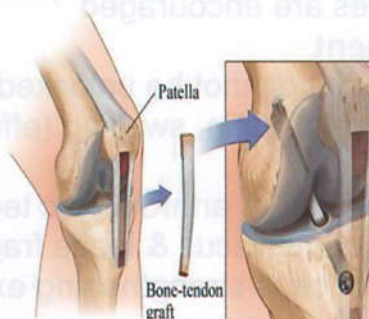
B) Surgical treatment

-Indication:

- In ACL injuries (**because of the instability they produce**) when conservative treatment fails, particularly in athletes who require full joint function.

-Operation:

- Reconstruction using a segment of another larger ligament to replace the torn ACL.
- The commonly used is the patellar ligament (about one-third of this ligament is removed & is secured to the femur and tibia to replace the torn ACL)
- It is not possible to repair the torn ACL by simply reconnecting the torn ends.
- Postoperative physiotherapy is essential.



-2 sets of ligaments present in the knee → cruciate & collateral ligaments

Cruciate ligaments

- Site: inside the knee joint
- Function: act as short ropes that connect femur to tibia & hold the knee joint tightly in place with flexion and extension → proper knee joint movement.

- ACL

- Site: front of the knee
- Function: prevents excessive forwards sliding of tibia beneath femur

- PCL

- Site: back of the knee
- Function: prevents excessive backwards sliding of tibia beneath femur

Fractures of the shaft of the tibia

Fractures of the tibia and fibula

Trauma

1- Direct violence

- As in RTA
- Fractures occur at same level in two bones & commonly transverse
- Usually compound and there is often significant displacement.

2- Indirect violence due to torsion or bending injury.

Complications

1- Skin damage

- As the tibial shaft is directly subcutaneous → tibial fractures are usually compound.
- Early skin closure by skin flaps is vital for healing and prevents infection.
- External fixation of the fracture is recommended.

2- Vascular injuries

3- Malunion.

4- Delayed and non-union (in up to 20% of cases)

5- Infection of compound fractures.

6- Stiffness of the ankle and subtalar joints

Treatment

1-Closed reduction and plaster fixation

- Indications:** a) for patients < 16 years old
b) Stable reduction in older patients

2- Skeletal traction

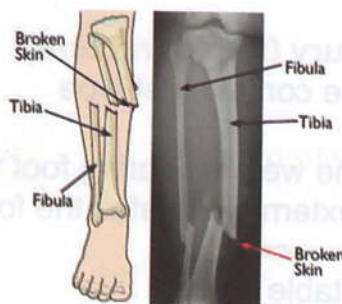
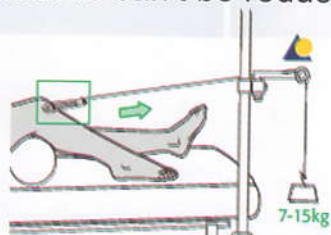
- **Method:** through a pin in the lower end of tibia or calcaneus
- **Indication:** cannot maintain stable reduction by plaster alone

3- External skeletal fixation

- **Method:** Special pins are fixed in the tibia above and below the fracture and then attached to special external fixators
- **Indication:** For compound fractures of the tibia as it allows:
 - a) Proper reduction
 - b) Leaves the wound exposed for dressings, skin grafts or skin flaps

4- Internal fixation

- **Method:** with a compression plate or an intramedullary nail.
- **Indications:** a) multiple fractures
b) In patients whom fractures can't be reduced by closed methods



Injuries of collateral ligaments

- Trauma:

- a) Valgus knee strain → medial collateral injury
- b) Varus knee strain → lateral collateral injury

- **C/P:** Pain, swelling, tenderness and excessive valgus or varus movement

- Investigation:

MRI (diagnostic)

- Treatment:

Conservative (similar to that for meniscus)

Isolated fractures of the tibia

- Trauma:

Torsion → spiral fracture with slight displacement.

- Treatment:

conservative by an above-knee plaster cast.

Ankle fractures

Trauma

1- Indirect violence

-The foot being either externally rotated, internally rotated (less frequent) on the tibia, inverted (adducted) or everted (abducted)

2- Direct violence:

- a) **Axial loading** → drives the talus vertically up into the tibia.
- b) Force which shears the talus transversely relative to tibia (rare)

External rotation injury (Pott's fracture)

- Incidence: This is the commonest type

- Trauma:

-A sudden arrest of the weight bearing foot whilst the tibia continues to move forwards → externally rotates the foot on the tibia

- Types:

Type I (the ankle is stable)

- The talus pushes the lateral malleolus posteriorly → rupture of the anterior inferior tibiofibular ligament
- Followed by an oblique (**spiral fracture**) of the distal fibula.
- The posterior inferior tibiofibular ligament and the medial structures of the ankle are still intact.

• **Type II** (the ankle is not stable), in addition to type I:

- If the force continues to act, the medial structures fail either by:
 - a) Rupture of the deltoid ligament
 - b) Avulsion fracture of the medial malleolus.
- Lateral dislocation of the talus may occur.

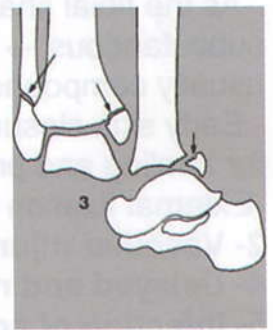
• **Type III**

- **Fracture third malleolus**

(The rotating talus → fracture of a small malleolar fragment from the posterior aspect of the distal articular surface of the tibia)

- Further posterior dislocation of talus will occur

During normal walking the feet are placed in a little external rotation relative to the line of travel



Complications

1- Malunion

-Results from failure to reduce and stabilize unstable fractures.

2- Non-union (occasionally occurs)

3- Ankle osteoarthritis (articular surface is not smooth) due to inaccurate reduction

4- Ankle stiffness

Treatment

A) Stable fractures

- Application of a weight bearing below-knee plaster cast for **6 weeks**

B) Unstable displaced fractures

Accurate open reduction and internal fixation

BEFORE

AFTER



Infections of bones and joints

Acute haematogenous osteomyelitis

Type of patient

- Almost a disease of children.
- It may even affect the neonates, particularly premature babies

Etiology

Causative organisms

- 1- Staphylococcus aureus (the commonest organism → **80%**)
- 2- Other Gram +ve cocci & Gram -ve bacteria (less common)

Source

Infection is blood borne from a septic focus e.g. a boil

Pathology

Site

- Most common in the metaphysis of long bones (often at the lower end of femur and upper end of tibia)
- This predilection for the metaphysis is due to:
 - 1- The metaphysis are supplied by end arteries in children → Bacteria or emboli are easily trapped → infarction.
 - After epiphyseal fusion, vascular communications are formed between metaphyseal & epiphyseal arteries → metaphysis contains no more end arteries → no longer subject to osteomyelitis
- 2- The metaphysis is the part most prone to trauma.
- 3- It is the actively growing end of bone.
- 4- The metaphysis is the part subject to strain (due to ligaments and muscles attachment)

Pathogenesis

- Fate: either (a) Resolution (b) Chronic osteomyelitis
- The characteristic pattern: a sequence of inflammation, suppuration, necrosis, reactive new bone formation and resolution & healing

1. Inflammation and suppuration (the earliest change)

- An acute inflammatory reaction → vascular congestion & exudation
- By 2nd or 3rd day, pus forms within the bone → intraosseous pressure rise rapidly → intense pain & obstruction of blood flow.

2. Extension and bone necrosis (sequestration).

a) Horizontal spread

- Results in subperiosteal abscess → obliteration of periosteal vessels & formation of small cortical sequestra (dead bone)
- Later, abscess ruptures → open on the skin → discharging sinuses.

Types of bone infections

A) Acute bone infections

1. Acute hematogenous osteomyelitis.
2. Acute osteomyelitis 2^{ry} to open fractures.
3. Acute osteomyelitis in association with orthopaedic Implants (as after internal fixation of fractures or joint replacement)

B) Chronic bone infections

1. Chronic osteomyelitis as a sequel to acute osteomyelitis
2. Brodie's abscess.
3. Chronic sclerosing osteomyelitis (**Garre's type**).

- Route of Infection:

- a) Blood stream from a distant site
- b) Direct invasion from a skin puncture, operation or an open fracture

b) Vertical spread

- Along the medulla → thrombosis of the major nutrient vessels and massive sequestration of the shaft.

- The epiphyseal cartilage prevents spread of infection to the joint.

- Sequestrum appear in X-ray dense white & if separation occurs, it appears as a black hollow surrounding the sequestrum

3-New bone formation, at two sites:

a) The deep layers of the stripped periosteum lay down bone by the end of **2nd week**, the new bone thickens → an involucrum formed enclosing the infected tissue and sequestra.

- If infection persists, pus discharges through a perforation in the involucrum (cloaca) & a track to exit at a sinus opening on skin surface; now condition is established as chronic osteomyelitis

b) Around the Haversian canals → bony sclerosis.

4. Resolution

- Bone around zone of infection is at 1st osteoporotic (due to hyperaemia)

- With healing, there is fibrosis & oppositional new bone formation, together with the periosteal reaction → sclerosis & bone thickening

- Remodeling may restore the normal contours; in others healing is improper → permanent deformity.

- The sequestrum is dead bone.
- Chronic osteomyelitis is rare nowadays due to:

a) Use of antibiotics

b) Early release of increased intraosseous pressure

Complications

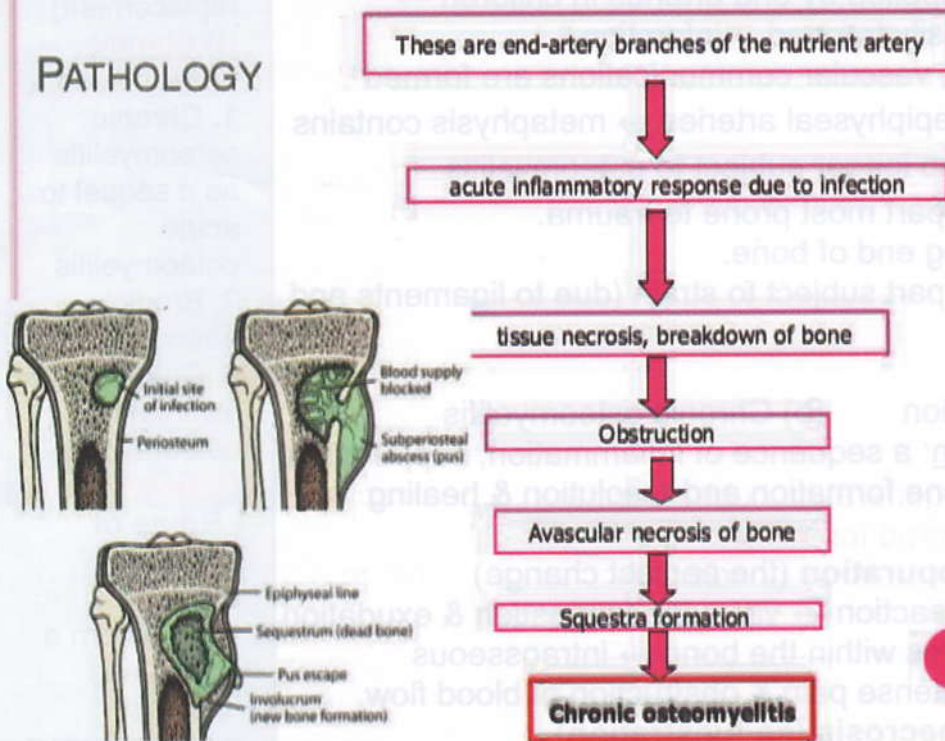
Systemic

Metastatic infection (pyaemic abscesses)

Local

1. Chronic osteomyelitis.
2. Suppurative arthritis (Epiphyseal cartilage prevents spread of infection to the joint)
- Suppurative arthritis occurs only in joints where part of the metaphysis is intracapsular as shoulder joint.
3. Altered bone growth.
4. Pathological fracture

PATHOLOGY



Clinical picture

Symptoms

- 1- A child with severe limb pain, malaise and fever.
- 2- The child is afraid to use the limb.

Signs

- 1- Severe localized tenderness over inflamed bone (**earliest sign**)
- 2- Later → warmth, edema and redness of the skin
- 3- The neighbouring joint can be moved passively with gentleness but painful
- 4- Effusion in the neighbouring joint

Investigations

A) Laboratory

- 1- Polymorphonuclear leukocytosis and raised ESR.
- 2- Blood sample for culture (taken before starting antibiotic therapy)

B) Radiological

- 1- Aspiration of pus (**the most certain to confirm the diagnosis**)
 - From the metaphyseal subperiosteal abscess or the adjacent joint.
- 2- MRI
 - Detect osteomyelitis before radiological signs appear on plain X-ray
- 3- Isotope scan with $^{99m}\text{TcHDP}$ (hydroxymethylene diphosphonate)
 - In very early stage → increased activity (relatively low specificity)
- 4- X-ray
 - During the 1st **two weeks** → normal
 - Later → rarefaction & periosteal new bone formation (treatment should not be delayed while waiting for these signs to appear)

Treatment

A) General management

- 1- Supportive treatment for pain and dehydration
- 2- Splinting of the affected part until the inflammation subsides and X-ray shows that there is no risk of a pathological fracture.

(Full weight bearing is usually possible after **3-4 weeks**)

B) Antibiotic therapy (**the core of treatment**)

- Time of administration: early after taking a blood or abscess sample for culture (Antibiotic may be changed according to culture results)
- Dose: high-dose IV
- Antibiotics used are effective against *S. aureus* as:
 - 1- Clindamycin
 - 2- First-generation cephalosporins
 - 3- Oxacillin
- Vancomycin is administered for MRSA.
- After about **5 days** when patient's condition improves → shift to oral antibiotics for **3-6 weeks**.

C) Surgical drainage

- Indication: no good response to antibiotics within **2 days** (This denotes the presence of pus which should be drained)
- Method:
 - 1- General anaesthesia
 - 2- Explore the inflamed area, open the periosteum → evacuate pus → send for culture & sensitivity (Some surgeons advise drilling of the cortex to evacuate any pus in the medullary cavity)
 - 3- The wound of the skin is loosely closed

Differential diagnosis

1. Cellulitis

(In osteomyelitis tenderness is elicited along the bone away from the area of redness)

2. Acute Suppurative arthritis

(Tenderness is over the joint & all movements "active & passive" are painful and limited)

3. Rheumatic arthritis.

- The pain is fleeting from one joint to another and patient may have other manifestations of rheumatic fever

4. Hemarthrosis

5. Ewing's sarcoma



Chronic non-specific osteomyelitis

Etiology

The causative organisms

-Usually a mixed infection (Staphylococcus aureus, E. coli, Strept. pyogenes, Proteus and Pseudomonas).

Route of infection

- 1- Follows an open fracture or operation (in presence of implants)
- 2- Blood spread as a sequel to acute osteomyelitis

Clinical picture

Symptoms

- 1- History of acute osteomyelitis may be given.
- 2- Pain and swelling in the affected bone
- 3- A sinus discharging pus (**the commonest presentation**)

Signs

- 1- Atrophy of the surrounding muscles
- 2- The affected bone is thickened, tender with sinuses that are lined by red granulation tissues, exuding pus
- 3- When the sinus is probed, the probe reaches the bone

Investigations

A) Radiological

1- Plain x-ray (**the most important**)

- It shows thickened bone with patchy & irregular sclerosis surrounding a bone cavity that may contain sequestra.
- Sequestra appear as dense white loose fragments with irregular but sharply demarcated edges.

2- Radioisotope scanning

- Using technetium (^{99m}Tc)_HDP scans, gallium citrate & indium (^{111}In)-labelled leukocytes → show up hidden foci of infection.

3- CT and MRI

- Show extent of bone destruction & reactive edema, hidden abscesses & sequestra → valuable in planning operative treatment

B) Laboratory

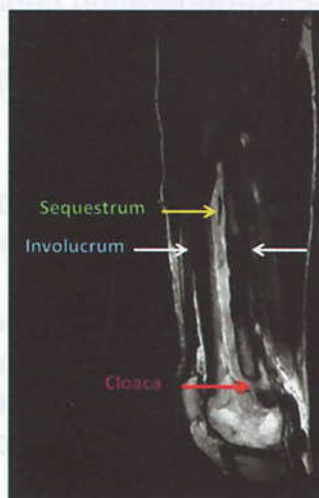
- ESR, CRP and WBCs are elevated during acute flares

Factors that maintain chronicity

1. Bone cavity surrounded by dense sclerosis
2. Sequestrum (dead bone), which acts as an irritant & harbours bacteria
3. Bacteria are imprisoned in the fibrous tissue (remain dormant & may be activated at any time)
4. Sinuses → open on skin surface → favour 2^{ry} infection

Complications

1. Acute exacerbation.
2. Pathological fracture.
3. Retardation or arrest of growth when the disease destroys the epiphyseal cartilage
4. Toxaemia & amyloidosis



Treatment

Surgery (the main line of treatment)

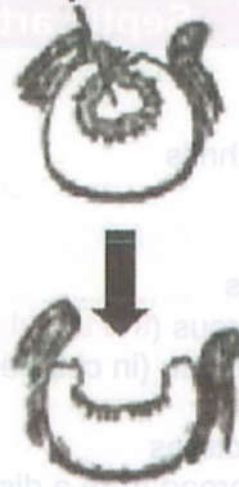
- Operation:

- Sequestrectomy → removal of dead bone.
- Saucerization of a bone cavity → provides adequate drainage.
- Obliteration of the dead space by cancellous bone chips or local muscle flaps → enhances healing.

- Postoperative

- Support of the limb to avoid a pathological fracture until healthy strong bone regenerates.

sequestrum



Antibiotics

- Are of little value if used alone because they cannot reach bacteria hidden in avascular tissue & sequestra
- They benefit in:
 - An acute exacerbation
 - When given with operative drainage

Brodie's abscess

Definition

A chronic bone abscess

Incidence

- Commonly affects adolescents
- Sometimes adults with high resistance → localization of infection

Pathology

- **Causative organism:** Staph. Aureus or albus
- **Size:** usually of small size,
- **Site:** in the metaphysis of long bone (commonly upper end of tibia)
- The abscess cavity may be sterile.
- The abscess is surrounded by dense sclerosis and there is always an associated periosteal reaction

Clinical picture

Symptoms: Recurrent attacks of pain in the affected area.

Signs: 1-localized swelling and tenderness over the bone
2- Effusion may be present in the adjacent joint

Investigations

X-ray → a translucent area surrounded by sclerosis



Treatment

Surgery

- The cavity is unroofed (under antibiotic coverage)
- Pus is evacuated then curettage
- Packing with cancellous bone chips (if the cavity is not small)

Septic arthritis

Definition

Acute Suppurative arthritis

Incidence

Common in children

Etiology

Causative organisms

- 1- Staphylococcus aureus (**the usual organism**)
- 2- Haemophilus influenzae (in children **< 3 years** of age)

Routes of infection

- 1- Following joint punctures
- 2- Haematogenous spread from a distant site
- 3- Direct extension from a nearby infection such as osteomyelitis
(If the metaphysis is intracapsular as in shoulder joint,
Otherwise the epiphyseal cartilage prevents spread to the joint)

Complications

- 1- Chronicity
- 2- Pathological dislocation
- 3- Secondary osteoarthritis
- 4- Pyaemia.

Clinical picture

Type of patient: More frequent in childhood

Symptoms

- 1- Pain (later become throbbing)
- 2- Swelling

Signs

-**General:** manifestations of toxæmia

-**Local:** 1- There may have been a wound.

2- There is deformity, redness, tenderness and limitation of joint movements equally in all directions.

3- Muscle spasm → keeps the joint in the position of greatest ease
(Position is variable from one joint to another depending on the relative strength of antagonistic muscle groups)

Investigations

A) Laboratory

High leucocytosis and elevated ESR

B) Radiological

1- Ultrasound guided joint aspiration

The aspirated fluid is rich in pus cells → send for culture & sensitivity to reveal the causative organism.

2- X-ray

- In early cases (**first 2-3 weeks**) → no abnormalities detected
- Later, there is: a) Rarefaction of the articular bone ends
- b) Narrowing of joint space
- c) Irregularity of joint surfaces
- d) Subchondral sclerosis

Pathology

-**Site:** commonly in knee & hip joints (any joint can be infected)

-**Pathogenesis:**

1. Organisms 1st settle in the synovial membrane (synovitis) → becomes red, swollen and exudes pus.
2. The infecting organism produces lytic enzymes → destroys articular cartilage & bone ends exposes
3. Pus bursts out of the joint → abscesses and sinuses.
4. With healing, the raw articular surfaces adhere → fibrous or bony ankylosis
5. If treatment begins early (at synovitis stage before articular cartilage damage occurs) → Recovery of joint mobility can be achieved

-Therefore, septic arthritis is **A surgical emergency**

Differential diagnosis

1-Acute osteomyelitis

- a) The tenderness is maximal over the bone not the joint
- b) A small range of joint movement is permitted.

2- Rheumatic fever

- a) Is polyarticular & fleeting with less severe general manifestations
- b) Relieved by large doses of salicylates

3-Haemarthrosis

There is history of trauma and aspiration reveals blood.

4- Tuberculous arthritis

- a) Insidious onset and chronic course.
- b) A cold abscess may be present
- c) Muscle atrophy is marked
- d) X-ray shows rarefaction of bone ends

Treatment

Septic arthritis is a surgical emergency.

General

- 1- IV antibiotics
- 2- Splinting until inflammation subsides, Later → gradual physiotherapy
- 3- If the articular cartilage has been destroyed → the joint is immobilized in the optimum position of function until ankylosis starts

Specific

-Early drainage by either arthroscopy or open arthrotomy

Acute osteomyelitis & septic arthritis

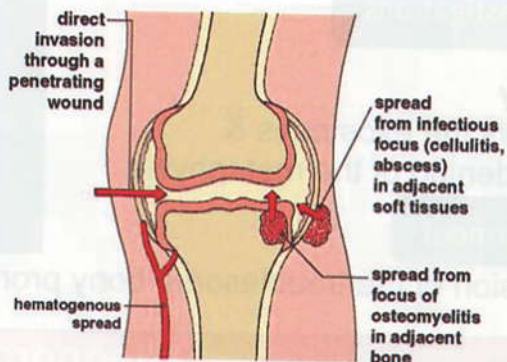
-Type of patient:

commonly in children (may even affect neonates)

- Treatment:

need **urgent** treatment.

- a) High-dose of IV antibiotics are essential
- b) Urgent drainage if pus is formed



Generalized disorders of bones and joints

Metaphyseal aclasis (Hereditary multiple exostoses)

Pathology

- 1- The disease is inherited as an **autosomal dominant** trait
- 2- Characterized by:
 - a) Appearance of multiple exostoses at metaphysis as child grows
 - b) Each exostoses covered by cartilaginous cap (**osteochondromata**)
 - c) Exostoses grows only while the child grows, any later enlargement → suspicious of malignant change to chondrosarcoma

Clinical picture

Symptoms: multiple painless swellings

Signs:

- 1- The child may be slightly short
- 2- Mild or moderate bowing of the arms or legs.
- 3- Occasionally, the bony prominences may cause:
 - a) Pressure to a neighbouring nerve or blood vessels
 - b) Interfere with the movement of a joint



Investigations

X-ray

Shows the exostoses & broadening of the metaphysis



Treatment

Excision of the troublesome bony prominence

Bone dysplasias

(Or faulty development)

- Definition: a generalized disorder of bone and cartilage
- Incidence: Uncommon
- The diagnosis is often difficult.
- Etiology: Most dysplasias are familial & patients are short → categorized as dwarfs (**<125 cm**)
- Commonest type: Metaphyseal aclasis (hereditary multiple exostoses)

Osteoporosis

Definition

Reduction in the amount of bone substance in the skeleton

Etiology

A) Primary (idiopathic) osteoporosis

Risk factors include: 1- Old age.

- 2- Female gender
- 3- Sedentary lifestyle.
- 4- Menopause before age 45.
- 5- Tobacco and alcohol excess.
- 6- Fair skin.



Normal

Osteoporosis

B) Secondary osteoporosis

- 1- Hyperparathyroidism
- 2- Hyperthyroidism
- 3- Cushing's syndrome and long-term steroid therapy
- 4- Calcium, vitamin D, or vitamin C deficiency

Clinical picture

- 1- The disease itself is usually symptomless.
- 2- Repeated spine minor fractures → chronic back pain and kyphoscoliosis
- 3- Osteoporosis leads to a clinical problem when a fracture occurs

Investigations

1- Dual-energy x-ray absorptiometry (DXA or DEXA scan).

- The most widely used technique for assessing bone mineral density

2- Plain X-ray (not sensitive)

- Shows decreased bone density when bones lose 40-50% of their substance

Prevention

- 1- Balanced diet
- 2- Calcium & vitamin D supplements for menopausal women.
- 3- Regular exercise as walking.
- 4- Avoidance of alcohol and smoking

Treatment

A) Primary osteoporosis

- 1- Calcium 1500mg/day.
- 2- Vitamin D 400 U/day.
- 3- Fluoride
- 4- Calcitonin: reduces osteoclastic activity → bone resorption reduction
- 5- Biphosphonates → osteoclastic activity
- 6- Hormone replacement therapy for menopausal women.

B) Secondary osteoporosis → treatment of the cause

C) Treatment of complications

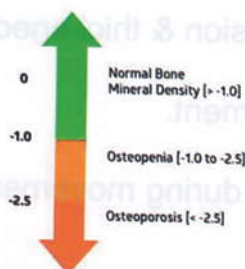
Fractures are treated according to their locations and types

Pathology

- 1-Normally the bone mass diminishes slowly but steadily from the age of 40 years (markedly accelerated around the menopause)
- 2- The amount of bone substance shows generalized reduction in the whole skeleton → reduced bone density → fractures after trivial trauma
- 3-Histologically, bone trabeculae appear thin with wide marrow spaces.
- 4- There are no demonstrable metabolic changes

Fractures commonly associated with senile osteoporosis:

- a) Colles' fracture
- b) Fractures of the vertebral bodies
- c) Fracture of femoral neck



Osteoarthrosis (osteoarthritis)

Incidence

A common degenerative joint disease

Etiology

A) Primary osteoarthrosis → no obvious cause.

B) Secondary osteoarthrosis follows such abnormality as:

- 1- Trauma
- 2- Deformity
- 3- Avascular necrosis (**Perthes' disease**)
- 4- Torn meniscus

Pathology

- Pathogenesis: disparity between stresses applied to articular cartilage & ability of cartilage to withstand such stresses → Structural damage

- The cardinal features are:

- 1- Progressive cartilage destruction → cartilage surface is roughened (**Fibrillation**) → extends progressively into deeper tissue → bone is exposed (**ulceration**)
- 2- Subarticular cyst formation and sclerosis.
- 3- Remodeling of the bone ends and osteophytes formation at the unstressed areas
- 4- Low grade inflammatory response → Capsular fibrosis

Clinical picture

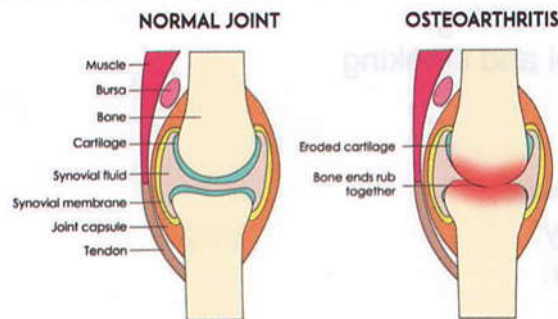
Type of patient: usually after middle age

Symptoms

- Pain and stiffness (the main symptoms)
 - o Pain increases by: a) movement b) change of weather
 - o Stiffness → marked in the morning & ↓ by the end of day
- Course: intermittent with periods of remission.
- Affected joints: weight-bearing joints such as the hips and knees

Signs

- 1- Joint swelling (due to effusion & thickened synovial membrane)
- 2- Tender joint line.
- 3- Restricted range of movement.
- 4- Deformity.
- 5- Coarse crepitus detected during movement.



Hyperparathyroidism

- There is generalized osteoporosis with thin cortex, deformity & probably bone cysts.
- In severe cases, osteoclastic hyperactivity → subperiosteal erosions, endosteal cavitation and replacement of the marrow spaces by vascular granulations & fibrous tissue (**osteitis fibrosa cystica**).
- Haemorrhage & giant cell reaction within the fibrous stroma → brownish tumor like masses → fluid-filled cysts "when liquefies" (**brown tumours**)
- C/P: bone aches and pathological fractures.
- Investigations:
 - a) **X-ray** → resorption of tooth sockets lamina dura & Subperiosteal phalangeal cortical erosions
 - b) **DEXA scan**: extent of Osteoporosis

Investigations

1-Plain X-ray reveals the characteristic changes:

- 1- Narrowing of the joint space.
- 2- Subarticular sclerosis.
- 3- Bone cysts.
- 4- Marginal osteophytes.



Treatment

A) Conservative treatment

-Indication: in early stages.

- Methods:

- 1- **Load reduction**, can be achieved by:
 - i) A walking stick
 - ii) weight reduction
 - iii) Avoidance of prolonged stressful activity.
- 2- **Pain relief** by analgesics and anti-inflammatory agents.
- 3- **Physiotherapy**

To overcome contractures & strengthen surrounding muscles → improve Joint mobility

B) Surgical treatment

Total joint replacement

-Indication: failure of conservative treatment to control symptoms

-Replacement of the hip and knee are commonly performed operations that allow patients to regain painless joint movement



Sclerosis & osteophytes distinguish osteoarthritis radiologically from rheumatoid arthritis (in which the predominant radiological feature is bone loss)



Charcot osteoarthropathy (neuropathic arthritis)

Pathology

- 1- Joint lacking deep sensations → rapidly progressive degeneration
- 2- Repeated minor injuries → severe disorganization of affected joint

Etiology

- 1- **Diabetic peripheral neuropathy** (the commonest cause)
- 2- Meningomyelocele
- 3- Spinal cord injury
- 4- Leprosy
- 5- Syphilitic tabes dorsalis.

Clinical picture

Symptoms

1- Painless joint swelling

2- Deformity and instability

Signs

1- Joint effusion

2- Abnormal joint mobility.

3- Signs of accompanying neuropathy are present, as:

a) Anaesthesia

b) Lost tendon jerks

c) Penetrating ulcers of the foot

Investigations

X-ray, shows:

1- Gross bone destruction

2- Widening of joint spaces

3- Joint is often subluxated or dislocated

4- Irregular calcified masses in the capsule

(sometimes seen)



Treatment

A) Conservative treatment

- Indication: applied to the majority

- Method:

1- Joint stabilization by a padded cast → minimize deformity

2- A special footwear and reduction of weight bearing

B) Surgical treatment (infrequently required)

- Indication: severe deformities that render the limb useless

- Operation: Amputation and fitting prosthesis

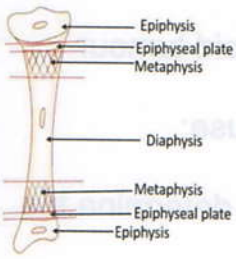
The underlying condition may need treatment, but the affected joints cannot recover

BONE TUMOURS

Classification

Cell type	Benign	Malignant
Bone	Ivory osteoma Osteoid osteoma Osteoblastoma	Osteosarcoma
Cartilage	Chondroma Osteochondroma Chondroblastoma Chondromyxoid fibroma	Chondrosarcoma
Fibrous tissue	Non-ossifying fibroma (fibrous cortical defect)	Fibrosarcoma
Marrow (hematogenous)	Eosinophilic granuloma	Ewing's sarcoma Multiple myeloma Leukaemia Lymphomas
Vascular	Haemangioma	Angiosarcoma
Uncertain	Giant cell tumour	Malignant giant cell tumour

According to their site

Vertebrae	Flat bones	Long bones
Osteoid osteoma Osteoblastoma Haemangioma Myeloma Secondaries	Secondaries Myeloma Chondrosarcoma 	Epiphysis a) Giant cell tumor b) Chondrosarcoma Metaphysis → Osteosarcoma Diaphysis a) Secondaries b) Ewing's sarcoma

Clinical picture

Type of patient (Age is a useful clue)

- 1- Childhood and adolescence → a) many benign lesions
b) Some 1^{ry} malignant tumours as osteosarcoma & Ewing's tumour
- 2- In older people (40-60 years) → Chondrosarcoma & fibrosarcoma
- 3- Above age of 60 years → Myeloma (the commonest 1^{ry} malignant bone tumors)
- 4- Above 70 years of age → metastases commoner than 1^{ry} tumours

General Principles

- Tumours, tumour-like conditions and cysts have the same clinical Presentation & management
- Bone tumours vary in morphology and biological potential.
- Most bone tumours are rare (0.5%).
- Benign bone tumours are common.
- 1^{ry} malignant bone tumours are rare.
- The most common malignant bone tumours are 2^{ry} metastasis.
- The second most common malignant tumours are haematogenous bone tumors
- Differentiation between benign and malignant bone lesions is not always easy

Symptoms

1- May be asymptomatic (discovered accidentally on X-ray or as a result of pathological fracture)

2-Symptomatic cases mostly will have pain & swelling

Signs: 1- Local tenderness 2- Pathological fracture

	Benign	Malignant
Age group	Young	Different
Course	Slow	Progressive
Pathological fracture	Rare	More common
Metastasis	No	Yes
General condition	Normal	Affected

Investigations

A) Laboratory

1-Biochemical test

2-tumour markers

B) Radiological

1- Plain X-ray (**the first line**)

i) May suggest the histological type of the tumour.

ii) The site of the tumour may suggest the diagnosis

2- CT scan and/or MRI (**the best investigations for staging**)

Assess lesion site, size & extension (osseous & extra-osseous)

3- Bone isotope scan → useful for detection of metastases

4-Angiography

C) Biopsy:

-Open biopsy performed after consulting radiologist & pathologist
- Done while a tourniquet is applied to the limb → minimize risk of tumour dissemination.

- Incision: longitudinal, through a muscle belly to avoid tumour spread through fascial planes.

- Site: the ideal part is the edge of the tumour, because:

1-The center may be necrotic

2- To obtain tumour & normal surrounding tissue to determine the microscopic extent of the tumour spread

- The biopsy site should be resected with the definitive surgery.

	Low grade tumour	High grade tumour
Differentiation	good	poor
Cellularity	Hypocellularity	Hypercellularity
Stroma	More	Minimal
Vascularity	Low	Hypervascularity
Necrosis	Minimal	Much

Lesions that simulate primary bone tumours on radiography

1. 2^{ry} tumours
 2. Osteomyelitis
 3. Callus from unnoticed stress fracture.
 4. Simple bone cyst.
 5. Aneurysmal small bone cyst.
 6. Brown tumour of hyperparathyroidism.
 7. Fibrous dysplasia.
 8. Myositis ossificans
- Tumours are classified as intraosseous, paraosseous, intraarticular or intramuscular (T staging)

-Definitive surgery should not be based on frozen section examination
- Histological grade is the most important feature of bone sarcomas and is essential for staging most of the bone tumour types

A) Surgery

1. Amputation (if possible)

The **main line** of treatment for many 1^{ry} malignant bone tumours

2. Limb preserving surgery.

- **Consist of:** tumour resection, skeletal reconstruction and soft tissue & muscle transfer.

- **The surgical guidelines and techniques are:**

- i) No major neurovascular tumour involvement.
 - ii) Wide resection of the affected bone with a normal muscle cuff in all directions
 - iii) En bloc removal of all previous biopsy sites and all potentially contaminated tissue.
 - iv) Resection of the adjacent joint and capsule
 - v) Adequate motor reconstruction (by regional muscle transfer)
 - vi) Adequate soft tissue coverage.
- Osteoarticular defects are reconstructed by segmental, custom prosthesis (fixed to the remaining intramedullary bone)
- Within the past few years, porous coating to the prosthesis has been applied → obtain biologic ingrowth → long- term fixation
- In addition Titanium devices have been introduced.

B) Radiotherapy

Uses:

- 1- **IN Ewing's sarcoma** as alternative to amputation to destroy the tumour (radiosensitive)
- 2- In combination with chemotherapy as adjuvant therapy and for inoperable lesions because of the size, local spread, metastatic deposits and marrow cell tumours as **myeloma and malignant lymphoma**.

C) Chemotherapy.

- The preferred adjuvant treatment with promising results as neoadjuvant therapy

- Drugs currently in use: 1- Methotrexate

2- Doxorubicin (Adriamycin)

3- Cyclophosphamide

4- Vincristine

5- Cis-platinum.

- The use of adjuvant chemotherapy has dramatically increased overall survival.

Treatment of bone tumours needs cooperation & consultation between the orthopaedic surgeon, radiologist & pathologist (in malignant Tumors → needed in addition the oncologist, prosthetic designer and rehabilitation therapist as well)

Solitary simple bone cyst

-Definition: a unilocular cavity filled with clear fluid

- Site: mostly in the metaphysis of the upper end of the humerus, femur or tibia, but other bones may be affected

- Diagnosis:

Type of patient: Young age (seldom seen in adults)

Symptoms: asymptomatic, usually discovered accidentally after:

a) A pathological fracture

b) An incidental X-ray finding as a well demarcated radiolucent area in the metaphysis with thinning out of cortex and bone expansion.

-Treatment:

1- Tends to heal spontaneously

2- If not → Injection of corticosteroids into the cyst (**80-160 mg** of methyl prednisolone) → lead to its obliteration.

3- If not → can be evacuated by scrapping the wall and filling the cavity with bone chips



Bone cysts

1. Simple bone cyst (solitary cyst).
2. Fibrous dysplasia.
3. Cysts associated with parathyroid osteodystrophy.
4. Hydatid cyst.
5. Aneurysmal bone cyst

Aneurysmal bone cyst

-Definition: A benign tumour-like lesion

-Site: any bone but mostly in the long bone metaphysis

-Incidence: at any age but more often in young adults

-Pathology: It contains cavities filled with blood and separated by connective tissue septa containing trabeculae of bone or Osteoid tissue and osteoclast giant cells.

- Malignant transformation does not occur.

-Investigation: X-ray →

1-A well-defined area that is trabeculated & eccentric

2- May resemble a giant-cell tumour.

-Treatment: by curettage and packing with bone chips.



Benign bone tumours

Osteochondroma (cartilage-capped exostoses)

Incidence

One of the commonest bone tumours

Pathology

1- **Site:** metaphysis of a long bone.

2- May be single or multiple (hereditary multiple exostoses)

3- Starts in adolescence, as a cartilaginous overgrowth at the edge of the epiphyseal plate → endochondral ossification → a bony protuberance still covered by the cap of cartilage.

4- The tumour continue to grow as long as the parent bone grows; any further growth → suggestive of malignant transformation

Clinical picture

- **Type of patient:** a teenager or young adult

- Symptoms

- 1- A painless lump
- 2- Pain (due to overlying bursa or pressure on soft tissue)
- 3- Parathesia (rarely, due to stretching of an adjacent nerve)

Complication: Malignant transformation, present by → pain, rapid growth, invasion of the mother bone & recurrence after excision

Investigation

X-ray

- Has a pathognomonic appearance → a well-defined exostoses emerging from metaphysis, its base is continuous with parent bone
- The cartilage cap is usually invisible → looks smaller



Treatment

If symptomatic → excised at its base

Giant cell tumour (osteoclastoma)

Pathology

Cell of origin

- Uncertain
- Derives its name from multinucleated giant cells, which are uniformly distributed in the tumour tissue.

Site

- Only in mature bone (those in which the epiphyses have fused)
- Common sites are **around the knee** (distal femur & proximal tibia) And **away from elbow** (proximal humerus & distal radius)
- It characteristically extends up to the subarticular bone without invading the articular cartilage

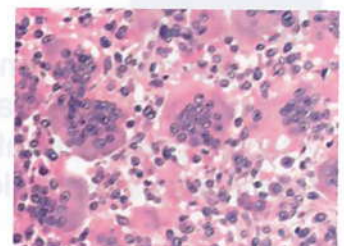
Gross picture

- Has a reddish brown, fleshy appearance
- It comes away in pieces quite easily when curetted, but difficult to remove completely from the surrounding bone.

Microscopic picture

- Abundant multinucleated giant cells scattered on a background of stromal spindle shaped or ovoid cells → responsible for determining the aggressiveness of the tumour.
- The numerical grading system (I, II and III) is considered a useful predictor of biological behavior.
- All giant cell tumours are considered potentially malignant

Giant cell tumours may present with Different grades of aggression, from totally benign to frankly malignant



Clinical picture

Type of patient usually a young adult (20-40 years of age)

Symptoms

- 1- History of trauma & pathological fracture in 10- 15% of cases
- 2- Pain at the end of a long bone
- 3- Slight swelling

Signs

- 1- A vague swelling of the end of a long bone
- 2- The consistency vary from soft, firm or an egg-shell crackling sensation (depending on the degree of thinning of expanded cortex)
- 3- The neighbouring joint is often irritated

Investigations

For diagnosis

X-ray, shows

- An eccentric radiolucent area at the end of long bone
- Bounded by the Subchondral bone plate.
- It has the characteristic soap bubble appearance (Due to reactive bone formation under the periosteum)
- Medullary plug (**operculum**) at junction of tumour with shaft

For Staging

1-CT scans and MRI

- Reveal the extent of the tumour, both within the bone and beyond.

2- Arthroscopy → detect the articular surface invasion

Treatment

Surgery (the main line)

-Methods:

1- Curettage and bone grafting (the simplest)

Disadvantage → recurrence is common.

2- Wide excision (the treatment of choice)

Replacement is done by specially designed prosthesis or by osteocartilaginous grafts

3- Amputation

Indication → recurring tumours with suspicious of malignancy

Radiotherapy → for surgically inaccessible tumors



Lack of medullary plug in imaging may signify either malignant osteoclastoma or bone secondaries



Primary malignant bone tumours

Osteosarcoma

Incidence

- It is the most common of primary tumours of bone
- It is a highly malignant tumour.
- Occurs predominantly in children and adolescents (Much less affects elderly people who have Paget's disease of bone)

Predisposing factors

1. Irradiation.
2. Paget's disease of bone.

Pathology

Cell of Origin → the primitive osteoblasts

Site → in the metaphysis of a long bone

Gross picture

- Osteosarcoma destroys and replaces normal bone.
- Characterized by the formation of bone or osteoid tissue
- Rapidly infiltrates the medulla towards the shaft but it respects the epiphyseal cartilage → not invade the epiphysis or the joint.
- There are four main pathological features:

- a) Bone destruction.
- b) Tumour bone formation (radiologically appears as **sunray**)
- c) Reactive bone formation (appears as **Codman's triangle**)
- d) Soft tissue infiltration.

- The tumour varies greatly in appearance. It may be:

- a) **Osteolytic type** → soft, fleshy and vascular with areas of haemorrhage and necrosis
- b) **Sclerosing type** → solid and contains bone

Microscopic picture

- Shows considerable variation between:
 - a) Areas having spindle cells with a pink-staining osteoid matrix
 - b) Others contain cartilage cells or fibroblastic tissue with little or no osteoid.
- The diagnosis depends on seeing evidence of osteoid formation

Clinical picture

Type of patient

- The highest incidence between the age of 10 and 20 years
- A second peak occurs after 50 years of age due to malignant changes in Paget's disease

Symptoms

- 1- Pain (the first symptom), characterized by:
 - a) Constant
 - b) Worst at night
 - c) Gradually increases in severity.

2- Swelling

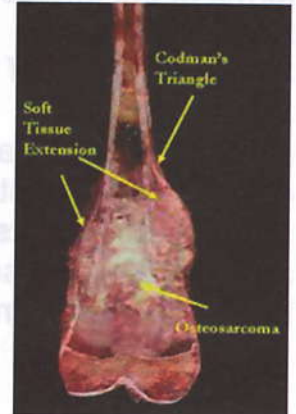
3- Pathological fracture (rare because the patient is bed ridden because of the pain)

Signs

- 1- Local tenderness (may be the only sign)
- 2- In later cases → a palpable mass and the overlying tissues may appear swollen and inflamed.
- 3- Enlarged regional lymph nodes

The rule of 80

- 80% in teenagers
- 80% in lower limb
- 80% around knee
- 80% in end of femur
- 80% in the metaphysis



Differential diagnosis

1. Chronic non-specific osteomyelitis.
2. Giant cell tumour.
3. Other malignant bone tumours as chondrosarcoma, fibrosarcoma, Ewing's sarcoma and reticulum cell Sarcoma
4. Secondary carcinomatous deposits.

Investigations

1-X-ray shows:

- Hazy osteolytic areas alternating with dense osteoblastic areas
- Often the cortex is breached and the tumour extends into the adjacent tissue → streaks of new bone formation appear, radiating outwards from the cortex → **sunburst (sun-ray) effect**
- Where the tumour emerges from the cortex, reactive new bone forms at the angles of periosteal elevation → **Codman's triangle**

2- CT and MRI

Show the extent of the tumour.

3- Chest X-ray →

may show pulmonary metastasis.

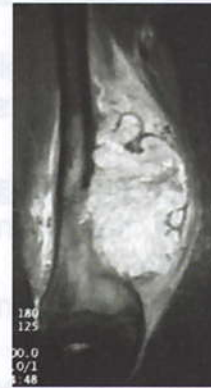
4- Biopsy → to establish the diagnosis.

5-Laboratory tests

- a) The ESR → raised
- b) Increased serum alkaline phosphatase level



Both sunray appearance and Codman's triangle are typical of Osteosarcoma, yet they may occasionally be seen in other rapidly growing tumours



Treatment

1- Local control of the disease is by either:

a) Amputation

The level should be proximal to the joint above the tumour, e.g., osteosarcoma at the tibia → an above knee amputation

b) Wide local excision and prosthetic replacement.

2. Adjuvant chemotherapy has markedly improved the prognosis

Chondrosarcoma

Definition

A malignant tumour characterized by the formation of cartilage

Incidence

- Highest incidence in the 4th and 5th decades
- Men are affected more often than women

Types

I) primary chondrosarcoma (a rare tumour)

- Rarely seen below the age of 40
- Occur in any bone that develops from cartilage, commonly in the pelvic bones, ribs or in the metaphysis of one of the tubular bones.
- It starts as a central (medullary) tumour similar to Chondroma and expands slowly

II) Secondary chondrosarcoma

- Secondary change in a pre-existing cartilaginous lesion most frequently in metaphyseal aclasis or multiple enchondromatosis
- Usually arises in the cartilage cap of an exostosis

Malignant change of osteochondroma is suspected by:

1. Progressive enlargement of osteochondroma after the end of normal bone growth.
2. Rapid increase in size.
3. Onset of pain.
4. Recurrence after local excision

Clinical picture

- 1-The tumour is slowly growing → a dull ache or a gradually enlarging lump
- 2-Medullary lesions may present as a pathological fracture

Investigation

X-ray → a destructive medullary tumour containing characteristic flecks of calcification



Treatment

Surgery

Chondrosarcoma metastasize late → at least one attempt at wide local excision is justified.

Radiotherapy → poor response

- Indication: when neither excision nor amputation is feasible

Ewing's sarcoma

Pathology

- A rare malignant tumour that arises from vascular endothelium in bone marrow
- Site: **diaphysis** of a long bone → give rise to a periosteal reaction
- Commonly affects tubular bones especially tibia, fibula or clavicle
- Spread: a) **Blood-born** metastasis to other bones and to the lungs
- b) **Lymphatic** spread to regional LNs is common

Clinical picture

May resemble those of osteomyelitis

Type of patient

Most commonly between the ages of 10 and 20 years

Symptoms

- Pain and swelling (the main presenting features)

Signs

General: 1) Intermittent or continuous pyrexia

2) Elevated ESR

Local: The swelling is warm & tender, ill-defined and is diaphyseal

Investigations

1-X-ray usually shows:

- a) **Onion peel effect** → an area of bone destruction (predominantly diaphyseal) with new bone formation in layers along the shaft
- b) **Sun-ray appearance and Codman's triangle** → due to tumor extension into surrounding soft tissues

Differential diagnosis

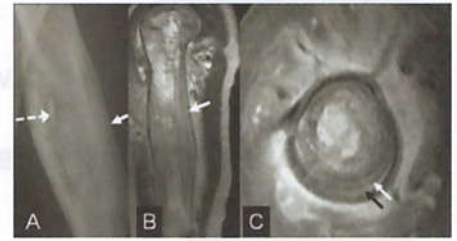
1. Secondaries from Neuroblastoma (Usually the patient is < 5 years of age)
2. Reticulum cell sarcoma. (Usually in patients > 20 years of age)
3. Osteomyelitis (Biopsy will establish the diagnosis)



2- Bone scans → multiple areas of activity in the skeleton

3-CT and MRI

Show extra-osseous component & local extent



Treatment

-The best results are achieved by combination of:

a) Chemotherapy b) Surgery c) Radiotherapy

Then a further course of chemotherapy for one year is given.

-The prognosis: poor despite the tumour is radiosensitive

Multiple Myelomas

Pathology

Cell of origin: plasma cells of the bone marrow

Site

Wherever red marrow occurs as in the trunk bones, the skull and proximal ends of femur and humerus

Microscopic picture

- Sheets of plasma cells.

- A plasma cell has a large eccentric nucleus that contains a spike-like arrangement of chromatin

Clinical picture

Type of patient: common between **45-65 years** of age

Symptoms

General: ill-health → Anaemia, cachexia and chronic nephritis

Local: 1- Weakness

2- Pathological fracture

3- **Bone pain** → is constant and backache is particularly common, sometimes with root pain and occasionally paraplegia

Investigations

Radiological → X-ray

1- Overall reduction of bone density

2- Sometimes there are multiple punched out defects with no marginal new bone round them (similar to metastatic bone lesions)

Laboratory

1. Urine analysis → Bence Jones protein

(**Coagulates at 55°C and disappears at 85°C**).

2. Plasma & urinary protein electrophoresis → characteristic pattern

3. Sternal puncture → reveals the typical myeloma cells

(Plasma cells form 10% or more of nucleated cells of the marrow)

4. Blood analysis → a) Anaemia b) Raised ESR

c) Raised alkaline phosphatase d) Elevated gamma globulin

e) Hypercalcaemia f) Plasma cell leukaemia

(where myeloma cells enter the circulation and elevate WBCs count)

Complications

1. Visceral involvement (liver, spleen and lymph nodes).
2. Myeloma kidney (blocking of tubules with protein casts).
3. Metastatic calcifications.
4. Amyloid disease



-Myeloma is one of the causes of secondary Osteoporosis
- (osteoporosis + a high ESR = myelomatosis) until proved otherwise

Treatment

A) Radiotherapy and chemotherapy

Relieve pain & pressure effects for a time and may prolong survival.

B) Surgery

Has a limited role for complicated cases.

C) Internal fixation → for pathological fractures in the limbs

D) Decompression → for unrelieved cord pressure

Differential diagnosis

1. Bone secondaries.
2. Parathyroid osteodystrophy
3. Osteoporosis

Bone Metastases

Incidence

More common than all primary bone tumors together

Pathology

Sources

-The commonest sources are carcinoma of the breast, prostate, kidney, lung, thyroid, and adrenal neuroblastoma.

-In about 10% of cases no primary tumour is found.

Site: Usually in areas containing red marrow.

Types

1. Osteolytic deposits

These destroy and replace bone by: a) their own expansion

b) Stimulating active bone resorption (osteolytic lesions) by:

(i) direct action of the tumour cells

(ii) tumour derived factors → stimulate osteoclastic activity

2. Osteoblastic (bone-forming) deposits are uncommon.

They usually occur in prostatic carcinoma

Metastases are more frequent than primary malignancies in:

- 1-Lungs
- 2- Liver
- 3- Bones
- 4- Brain

Clinical picture

Type of Patient: usually aged 50-70 years.

Symptoms

1-Pain (the commonest & often the only clinical feature)

2- A pathological fracture

3-Symptoms of hypercalcaemia:

a) Anorexia

b) Nausea

c) Abdominal pain

d) General weakness

e) Depression

f) polyuria

Signs

1- The primary tumour may be obvious

2- Examine the neck, breasts, axillae, lungs, abdomen and genitalia

3- Rectal or vaginal examination is necessary

Investigations

Laboratory tests

A- Low → haemoglobin

B- Elevated → 1- Serum alkaline phosphatase
2- ESR
3- Acid phosphatase & PSA (prostate specific antigen) in metastatic prostate carcinoma.



Imaging

1- X-ray

A) Osteolytic (most skeletal deposits) →

- appear as a **moth-eaten** appearance in the cortex.
- Sometimes there is bone destruction with or without pathological fracture.

B) Osteoblastic deposits → signify prostate carcinoma

2- CT scan

3- Radioscintigraphy with $^{99m}\text{TcHDP}$

- The most sensitive in detecting silent metastatic deposits in bone
- Areas of increased activity are selected for X-ray examination

Treatment

- Metastatic carcinomas usually have a bad prognosis
- **Treatment is palliative:**
 1. Painful deposits are treated by radiotherapy
 2. Pathological fractures are treated by a combination of internal fixation and radiotherapy.
 3. Chemotherapy



Deformities of bones and joints

Deformities of the elbow

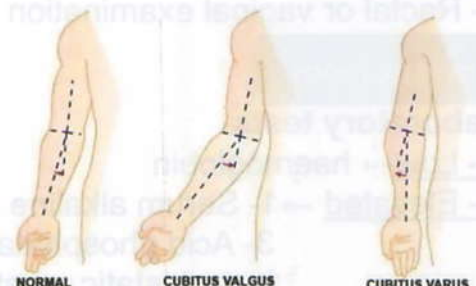
Cubitus valgus

- The carrying angle is increased → hand deviates away from body
- The most common cause is malunion of a fractured lateral condyle of the humerus.
- Clinical presentation: gross deformity & a bony knob on the inner side of the joint.
- Complication: delayed ulnar palsy → weakness of the hand with numbness and tingling of ulnar distribution (years after the injury)
- Treatment: 1- The deformity itself needs no treatment
2- For delayed ulnar palsy → transport nerve to front of the elbow

Cubitus varus

- The carrying angle is decreased → hand deviates towards body
- The most common cause is malunion of a supracondylar fracture of the humerus.
- Clinical presentation: The deformity is obvious when the elbow is extended and the arms are elevated and the hand brushes against the body in walking.
- Treatment: corrected by a wedge osteotomy of the lower humerus

Normally the forearm is in a position of slight abduction with the arm constituting an angle of about **10-15°** known as the **carrying angle**.



Deformities of the hip

Congenital hip dislocation (developmental hip dysplasia)

Etiology

- 1- Familial tendency
- 2- Intrauterine malposition has been blamed (have a higher incidence than of breech presentation)

Clinical picture

Mother's observations

- 1- Before walking → asymmetry, a clicking hip or difficulty in applying the napkins (because of limited abduction)
- 2- After walking → asymmetry more obvious & apparent limp

Examination

A) With unilateral dislocation

- 1- Asymmetrical skin creases on the two sides.
- 2- The affected leg is slightly short and rotated externally
- 3- **Trendelenburg's test** is always positive
(Standing on one foot, on the side of dislocated hip → pelvis drops on the unsupported (healthy) side "due to impairment of abductor muscles on the dislocated side")

B) Bilateral dislocation → more difficult to detect because:

- 1- There is no asymmetry
- 2- Characteristic waddling gait may be mistaken for normal toddling
- 3- Perineal gap is abnormally wide and abduction may be limited.

Investigations

1-Ultrasound scanning

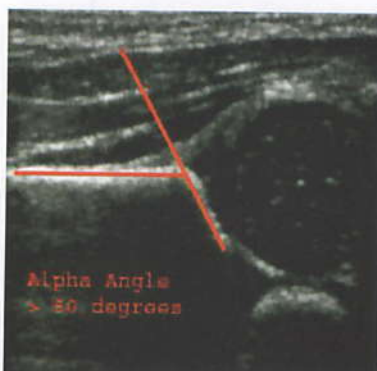
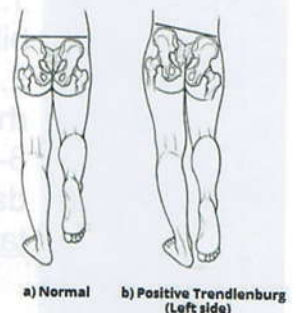
- Visualize the shape of acetabulum & position of femoral head with reasonable accuracy.
- Is becoming part of the routine assessment of all neonates.

2- X-ray examination

- a) In the newborn → of little value because head of the femur is entirely cartilaginous and not visible radiologically
- b) In a child of one year age or more it shows:
 - i) The acetabular socket is unusually shallow.
 - ii) The ossification centre of the femoral head is underdeveloped and is displaced upwards and outwards

Pathology

- 1- Acetabulum is unusually shallow
- 2- The femoral head slides out posteriorly.
- 3- The joint capsule though stretched remains intact and by folding inwards → hinder reduction
- 4- Maturation of the acetabulum and femoral epiphysis is retarded and the femoral neck is short & anteverted
- 5- Neglected cases → hip osteoarthritis in later years



Congenital hip dislocation should be diagnosed soon after birth when treatment is simple and most effective

Treatment

- Delay in diagnosis & treatment → worse prognosis.
- The principles of treatment → reduce the hip and to maintain reduction for an adequate period of time

In the neonatal period (up to 6 months)

- Most hips with +ve instability sign at birth → spontaneous healing
- If ultrasound scanning soon after birth shows that the acetabular roof is defective → start splinting using **von Rosen splint** for **3-6 months** by which time scanning should show a good acetabular roof.

Cases discovered between ages of 6 months - 2 years

- Closed reduction & fixation in a plaster cast with the hips abducted for **6 weeks** until acetabular development is satisfactory by serial x-ray → the plaster is then replaced by a splint

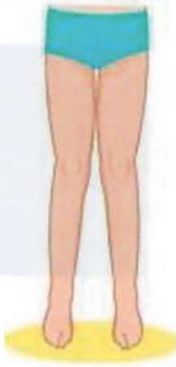
Children > 2 years

- Open reduction followed by fixation in a plaster cast.
- A markedly shallow acetabulum can be deepened by a concomitant pelvic osteotomy



Deformities of the knee

	Genu valgum (knock knees)	Genu varum (bow legs)
Definition	- Fixed abduction deformity of the knees from the midline. - When the knees are placed in contact → the malleoli separate from each other	While the knees are extended, there is lateral bowing of the legs → the knees are widely separated and the malleoli are in contact
Etiology	1. Idiopathic (the commonest & bilateral) 2. Bone softening (with rickets & rheumatoid arthritis) 3. Bone injury (with epiphyseal damage or following a fractured lateral tibial condyle)	1. <u>Physiological</u> (the commonest) Some babies are born with slight bow leg or develop it while wearing napkins → nearly all grow straight. 2. <u>Bone softening</u> due to rickets and Paget's disease 3. <u>Bone injury</u> affecting the epiphyseal site and sometimes following fracture of the upper tibia

Clinical picture	-The deformity appears when patient stands but disappears when knees are flexed. -Knees tend to knock together → patient walks with the knees partially flexed and separated and tends to fall during running <u>With idiopathic knock knee</u> 1. Deformity is the only symptom (appears at the age of 2-3 years & nearly recovers by the age of 6 years) 2-Other postural deformities such as flat foot may coexist but these children are normal in other aspects. • <u>In the other types</u>, the symptoms and signs of the underlying cause are present.  VALGUS Knock-kneed	-The bowing may involve the tibia alone or the femur may also be affected. -The deformity is ugly and causes the patient to look stunted.  VARUS Bow-legged
Treatment	<u>Of the idiopathic type</u> - Requires no treatment (reassure the parents & observe the condition) - If by the age of 3 years the intermalleolar distance is > 3 inches → do corrective osteotomy	-Idiopathic bow legs → correct spontaneously. - If bow legs persist after the age of 4 years → do Infracondylar tibial osteotomy

Deformities of the foot

Congenital talipes equinovarus (club foot)

Incidence

- Common deformity (1 in 1000 live births).
- Bilateral in 1/3 of cases

Etiology

- The cause is unknown but predisposing factors include
 - 1- Positive family history.
 - 2- Male gender (Boys are affected twice as often as girls)

Clinical picture

Symptoms

- 1- Deformity (the only symptom in infancy)
- 2- Painful callosities (develop years later in the forefoot if the deformity remains uncorrected)

- Signs**
- 1- The heel may be small & high
 - 2- the calf is thin.
 - 3- Gentle attempts at passive correction → fixed deformity
 - 4- Examine for associated disorders such as spina bifida.

Investigation

X-ray → shows the shape and position of tarsal ossification and is helpful in assessing the progress after treatment



Treatment

A) Non-operative treatment (manual correction).

- Treatment begins within 2 or 3 days of birth.
- Each component of the deformity is corrected in the following order:
 - 1st → forefoot adduction then inversion & finally the equinus.
- The foot gently manipulated without anaesthesia and held in position there by adhesive strapping or by a plaster cast.
- The process is repeated weekly for 6-8 weeks until the foot is not only corrected but overcorrected

B) Surgery

- Indications:

- 1- Resistant cases
- 2- Older children

- Procedure: includes

- 1- Reduction by elongation of tendo achilles & division of tight ligaments.
- 2- Osteotomies are needed for children > 5 years.
- 3- Maintaining reduction by casts then by splints.
- 4- Triple arthrodesis (for advanced cases)



Terminology

- **Talipes** → Ankle & foot
- **Equino** → Plantar-flexion of ankle that looks like a horse's hoof
- **Varus** → Inward turning of the foot (the sole looks inwards)

Pathological anatomy

The deformity includes three anatomical disturbances.

- 1- Planter flexion of the foot at the ankle.
- 2- Inversion of the calcaneus & the navicular on the talus
- 3- Adduction of the bones of the forefoot at the mid tarsal & tarsometatarsal joints



Flat feet (pes planus)

Etiology

Either: 1- Collapse of normal arches
2- Persistence of the infant shape.

Types

- 1- Congenital 2- Infantile 3- Spasmodic 4- Idiopathic

Idiopathic flat foot

Incidence

The commonest type of flat feet in adults and it may be

Etiology

The end result of repeated episodes of foot strain.

Clinical picture

Type of patient: commonly in people who spend long hours standing

Symptoms

- 1- Aching pain in the foot after long continued standing
- Course: gradually progressive
- Site: medial part of the sole (due to gradual collapse of medial longitudinal arch)
- Provoked by: dorsiflexion the forefoot → stretch plantar ligaments
- 2- In later stages, foot becomes painless and gait shuffling & inelastic

Signs

- 1- Local tenderness may be found in the sole under apex of the arch (because the plantar ligaments are overstretched)
- 2- The foot is rigid and flat.
- 3- All joints of the hind-foot degenerate.

Prevention

- 1- Arch supports
- 2- Exercises to improve the function of the intrinsic muscles of the sole and of the toe flexors

Treatment

Indication: symptomatic cases

Methods:

- 1- The pain of foot strain is relieved by rest.
- 2- When the foot collapses, little can be done to improve its appearance or function → advice patient to accommodate his life to his feet by taking a sedentary job.

- At birth the foot is flat & the normal arch is only acquired when the infant stands
- The arch is maintained in adult life by:
1. Shape of the bones of the foot
2. Connecting ligaments
3. Action of the muscles of the calf & sole.
- Foot strain is an ill-defined uncommon condition in which the foot becomes acutely painful following excessive use.



Hallux valgus

Definition

Outward deviation of the great toe at the metatarsophalangeal joint

Incidence

The commonest of foot deformities & all musculoskeletal deformities

Etiology

- 1- Badly fitting shoes with pointed fronts and high heels.
- 2- Congenital origin (rare)

Pathology

- 1- Outward deviation of the first toe at the metatarsophalangeal joint.
- 2- The deformity tends to be progressive.

3-Complications:

- a) Callosity in the overlying skin.
- b) Adventitious bursa over the prominent head → **bunion**
- c) Osteoarthritis of the metatarsophalangeal joint



Clinical picture

Type of patient:

-Age: Commonly in adolescents and in the 6th decade

- Sex: common in **females**

Symptoms

Features of complications as pain from a bunion

Signs 1- Hallux valgus is usually bilateral.

2- **Hammer toe deformity** → The big toe may lie above or below 2nd toe and the other toes are pushed laterally

3- The head of 1st metatarsal projects on the medial side of foot



Treatment

A) Conservative treatment

- Aim: relieving pain rather than correcting the deformity.

-Methods: wide shoes, toe spacers and bunion pads.

B) Surgery

- Indication:

Intolerable pain not responding to conservative measures

- Operation: Osteotomy → restore toe alignment.



Urinary tract diseases

Symptoms of urinary tract diseases

1-Pain

a) Renal pain

-Character: dull constant aching pain

-Site: in the renal angle (between the sacrospinalis & the last rib)

-Referral: to anterior renal point (at upper outer quadrant of abdomen)

-Cause: renal pelvis distension or to stretch of renal capsule.

b) Ureteric (renal) colic

-Character: colicky pain

- Site & Referral: extends from the loin to the groin.

-When the stone is just coming out of the kidney → localized to loin

-As stone moves downwards → pain felt in groin & may radiate to the upper thigh and to the scrotum in **males** or the vulva in **females**.

-When stone is impacted at lower end of ureter → symptoms of vesical irritation & pain referred to the tip of the penis.

- Associating symptoms: nausea and vomiting.

- Cause: acute obstruction by stone → distension & increased ureteric muscular contractions (peristalsis).

c) Vesical pain

-Character: varies from slight discomfort to severe agonizing pain with severe desire to micturate → expulsion of just a few drops of urine

- Site: in the suprapubic region especially with a full bladder.

- Referred to: the tip of penis.

- Causes: cystitis, bladder stone or bladder carcinoma.

d) Prostatic and seminal vesicle pain

-Character: deeply seated vague discomfort

-Site: in the pelvis

- Referred to: perineum, rectum or suprapubic region.

-Cause: usually due to inflammation.

e) Urethral pain

-Character: burning.

-Cause: urethritis (occurs during micturition)

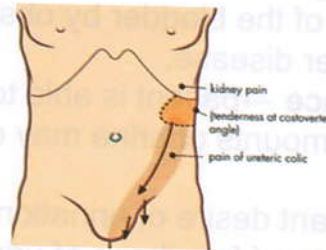
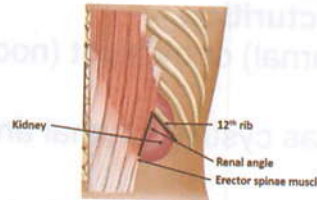
f) Testicular and epididymal pain

- Site: scrotum

-Radiation: to lower abdomen or costo-vertebral angle.

-Cause: epididymo-orchitis, trauma or torsion.

- Dull ache may be caused by varicocele.



Systemic symptoms

1. Symptoms of infection:

Fever, malaise & headache in **pyelonephritis** and

pyonephrosis

(Has rigors in addition)

2. Symptoms of metastasis:

Malaise, weight loss, back bony pain and symptoms related to involved organ

3. Symptoms of renal failure:

Malaise, nausea, vomiting, fatigue, forgetfulness, behavior changes, loss of weight, loss of libido, headache, pruritus and bleeding tendencies (as epistaxis and melena)

2-Symptoms related to micturition.

I) Symptoms of bladder irritation

-Causes: infections (as cystitis & prostatitis) or by a stone.

-These symptoms include:

a) Frequency of micturition.

- May be by day (diurnal) or by night (nocturnal).

- Causes:

(i) Bladder Irritation as cystitis (diurnal and nocturnal) & bladder stones (diurnal)

(ii) Mechanical reduction of bladder capacity, e.g., large bladder tumours or bladder fibrosis by TB, radiation or Schistosomiasis.

(iii) Functional reduction of bladder capacity as in patients with prostatic enlargement due to the presence of a big amount of residual urine (nocturnal frequency).

(iv) Polyuria

- **Definition:** Increased urine production by the kidneys

-**Cause:** excessive drinking or uncontrolled diabetes.

b) Urgency

-**Definition:** a strong sudden desire to urinate

- **Cause:** irritability of the bladder by obstruction, inflammation, or neuropathic bladder disease.

- **Urge incontinence** → patient is able to temporarily control urine but loss of small amounts of urine may occur

c) Strangury

-**Definition:** persistent desire of urination & straining at micturition with painful passage of few drops of urine that may be blood stained.

d) Dysuria

-**Definition:** painful urination, which is related to acute inflammation of the bladder, urethra, or prostate.

- **Associated symptoms:** urinary frequency and urgency

II) Symptoms of bladder outlet obstruction

a) Hesitancy and straining (the early symptom)

- **Definition:** Delay in initiating urinary stream

b) Difficulty in micturition.

In BPH, patient has to relax at beginning of micturition (hesitancy), while patients with urethral stricture have to strain to pass urine

c) **Loss of force & reduced caliber of the stream** → Noted with high urethral resistance despite increased intravesical pressure

d) **Terminal dribbling** → more noticeable as obstruction progresses

e) Interruption of urinary stream.

f) Sense of incomplete emptying

Due to presence of residual urine

g) Retention of urine acute and chronic

- The normal bladder capacity is about 400 ml.
- Prostate enlargement (benign or malignant) and urethral stricture are common Causes of bladder outlet obstruction

3-Urinary incontinence

4- Symptoms related to changes in urine

I) Changes in urine volume

a) Polyuria

-Cause: excessive fluids intake, DM, diabetes insipidus and high-output chronic renal failure.

b) Oliguria

-Definition: urine volume < 400 ml/24h in adults.

- Causes: i) Acute renal failure (due to shock or dehydration)
ii) Bilateral ureteric obstruction.

c) Anuria (one of urological emergencies)

- Definition: urine volume < 200 ml/24h.

-Causes:

i) **Pre-renal** causes as shock & dehydration.

ii) **Renal** causes as toxins, drugs & renal diseases.

iii) **Post-renal** causes as bilateral renal obstruction or complete obstruction in a solitary kidney.

II) Changes in color and content of urine

a) **Hematuria** → passage of blood in urine

b) **Pyuria** → presence of pus in urine (microscopic or macroscopic)

c) **Chyluria** → presence of lymphatic fluid or chyle in urine → appear white milky

- On adding ether → urine clears.

- On adding Sudan III → gives an orange coloration.

- The main cause: Filariasis

d) Cloudy (turbid) urine

Due to the presence of excessive phosphate crystals (phosphaturia) that precipitate in alkaline urine

-Adding few drops of acetic acid → clears the turbidity

e) Necroturia (pathognomonic of bladder carcinoma)

- Definition: Passing pieces of necrotic tissue in urine

f) Pneumaturia

- Definition: Presence of gas in urine

-Cause: mostly a fistula between urinary tract & colon that is caused by malignancy of colon or bladder or by colonic diverticulitis.

5- Urethral discharge (one of common complaints in men)

- Causes: urethritis, particularly the gonococcal type.

- Associated symptom: burning micturition

6-Bloody ejaculation (haemospermia)

- Cause: inflammation of the prostate or seminal vesicles.

7- Infertility

- Varicocele may reduce sperm count and motility.

-Mumps orchitis may cause testicular trophy.

8- Sexual difficulties in men.

- Erectile dysfunction, premature ejaculation, and loss of desire.

If Anuria is due to stones → called calcular anuria

- Infection can cause urine to be cloudy and malodorous

Examination for urinary tract diseases

A) General examination

- Signs of CRF may be present:

- 1- Pallor from anaemia
- 2- Eye puffiness
- 3- Dry tongue
- 4- Abnormal breath odour
- 5- Abnormal respiration
- 6- Drowsiness
- 7- Dehydration
- 8- Abdominal distension
- 9- Tremors
- 10- Convulsions
- 11- Oedema of lower limbs
- 12- Hypertension

B) Abdominal examination → may reveal a mass or fullness in renal angle or suprapubic area

C) Genital examination

• In males to look for: ulcers, meatal stenosis, hypospadias, epispadias, discharge, swelling, hydrocele and varicocele

• In females → vaginal examination

D) Digital rectal or bimanual examination (DRE)

To feel: 1- Anal sphincter tone. 2- Walls of anal canal & rectum

3- Prostate for size (**normal 4cm in length and width**), nodules, consistency (**normal rubbery**), tenderness and mobility

4- Seminal vesicles (normally not felt)

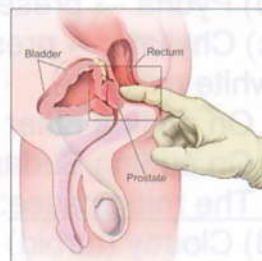
Shistosomiasis → calcification → palpable

5- Urinary bladder tumours are felt.

6- Malignant deposits in rectovesical or recto-uterine pouch

Differential diagnosis of a renal mass

1. Hydronephrosis
2. Pyonephrosis
3. Polycystic kidney.
4. Renal tumours
(**hypernephroma** or **Wilm's tumour**).
5. Big renal cyst



Investigations for urinary tract diseases

A) Laboratory

1- Urine analysis (mid stream) for

i) Physical properties

- Color: normally amber yellow.
- PH: normally acidic with a range 4.5-7.5.
- Specific gravity: normally 1005-1040 (according to hydration state)

ii) Biochemical analysis → for albumin, glucose and bilirubin

iii) Microscopic examination for

- Crystals as oxalates, uric acid, phosphates and cystine
- Casts as hyaline, granular and epithelial.
- Cells as RBCs or WBCs.
- Bilharzial ova and sperms.

iv) Microscopic staining for

- Pyogenic organisms by Gram stain.
- Acid-Fast (**Ziehl-Neelsen**) stain for TB.

v) Culture and antibiotic sensitivity.

vi) Cytological examinations for malignant cells

2- Urethral discharge analysis and culture

For gonorrhea & chlamydia

- Normal urine contain less than 5 RBCs & WBCs/HPF (high power field)
- Epithelial cells are present in normal female Urine

3- Renal function tests

- o Blood urea (N=15-40 mg/dL) (not an accurate test)
- o Blood urea nitrogen (BUN) (N=12-15 mg/dL) is more accurate.
- o Serum creatinine (**the best**) (N=0.2-1.5 mg/dL).
- o Creatinine clearance test (N=70-140 mL/m).
- o Serum electrolytes (serum K⁺ is elevated in renal failure)

4- Serum PSA (prostatic specific antigen)

For the diagnosis of prostate cancer (normally 4 ng/ml)

B) Imaging

1- Ultrasound

i) Abdominal & pelvic ultrasound

- **Advantages:** 1. simple, cheap and non-invasive technique.
- 2. Provides information about morphology, site & size of the kidneys as well as thickness of renal parenchyma.
- 3. Diagnose obstructive uropathy (**dilated pelvicalyceal system**)
- 4. Differentiate between cystic and solid lesions of the kidney.
- 5. Bladder tumours can be visualized.
- 6. Estimate the amount of post-voiding residual urine.

ii) Transrectal ultrasound

- Very accurate in diagnosing prostatic lesions.
- It allows guided biopsies from suspected prostatic cancer

2- Plain X-ray of urinary tract (known as plain UT or KUB)

- The following findings are looked for

- o Soft tissue shadow of the kidney (enlarged with renal swellings)
- o Psoas shadow (obliterated by a perinephric abscess)
- o Bony skeleton (Look for spina bifida, wide separation of symphysis pubis → in cases of ectopia vesicae, fractures & bone metastases)
- o Radio-opaque shadows:

- i) Urinary stones (renal, ureteric, bladder and prostatic).
- ii) Abnormal calcification of UT as that of renal TB & renal tumours
- iii) Bilharzial bladder & calcified Bilharzial seminal vesicles (**honey comb appearance**).

iv) Calcifications outside the urinary tract → adrenal calcification, mottled calcified lymph nodes (**tabes mesenterica**), calcified iliac vessels and pelvic phleboliths

3- Intravenous pyelography (IVP) / intravenous urography (IVU)

- **Idea:** i) take X-ray films after IV injection of a radio-opaque (**iodine containing**) contrast material → **urograffin**.

- ii) The contrast is concentrated and excreted by the kidneys in urine.
- iii) Normally a nephrogram phase appears within a few minutes then pelvicalyceal system. Later, ureters & bladder are visualized
- iv) Afterwards the patient micturates and a post-voiding film is taken to evaluate residual urine, which is normally almost zero

- **Advantages:** 1-shows most of congenital, traumatic, inflammatory, and neoplastic lesions of the urinary tract.

2-Gives idea about functional capacity of the kidneys

-Creatinine clearance test means the amount of plasma completely cleared from creatinine by the kidneys in one minute (denotes qualitative kidney function accurately)

- Iodine contrast media are allergenic that's why precautions sensitivity testing should be done



4- Infusion urography

- Indication: patients with impaired renal function where IVU will not show up due to lack of secretion of contrast medium.

- Method: contrast material is mixed with saline and is infused slowly over a period of **15 minutes** to allow kidneys to concentrate contrast

- Contraindication: blood urea is >100 mg/dL

5- Retrograde (ascending) pyelography

- Indication: when data obtained from IVU are not conclusive or if IVU is contraindicated (e.g. uremia or contrast sensitivity).

- Method: cystoscope ureteric catheters are passed along the ureters to the renal pelvis and a contrast material is injected.

- Advantage: provides good delineation of the pelvicalyceal system but no information about the renal function



6- Cystography

- Types:

a) **Descending cystography** → During performing an IVU, the bladder will be visualized in late films but the pictures may not be of a high quality as the contrast is diluted with urine.

b) **Ascending cystography** →, a urethral catheter is passed to the bladder and **400ml** of saline mixed with contrast is infused

- Indication: identify bladder pathology or vesico-ureteric reflux



7- Urethrography

- Indication: diagnose urethral injuries, strictures or fistulous tracts

- Types:

a) **Micturating urethrography** during IVU.

b) **Ascending urethrography** by injecting contrast medium through external meatus

8- CT scan

- Uses: currently of choice for detection of urinary stones, staging of renal and bladder neoplasms and assessing urinary tract trauma.

- Can be supplemented with contrast injection

- Disadvantage: involve a high radiation dose.

9- Magnetic resonance imaging (MRI)

- Advantage: not involve radiation or iodide contrast media.

- Indications: staging of neoplasms, assessing venous involvement in renal cell carcinoma & evaluation of renal artery stenosis

10- Renal arteriography

- Indication: diagnosis of renal artery stenosis & renal tumours and for kidney donors before transplantation.

- Method: selective catheterization of the renal artery by a cannula through the femoral artery using the **Seldinger** technique

11- Radioisotope scanning

-Types:

a) DTPA (diethylenetetramine penta-acetic acid) → called **renogram**

- The isotope is rapidly excreted through the glomerulus.
- The radioactivity is traced by the use of a gamma camera.
- A triphasic curve is plotted describing:

i) Vascular phase in which the isotope reaches renal vasculature

ii) Secretory phase where it is filtered by the kidney

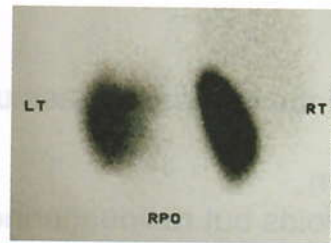
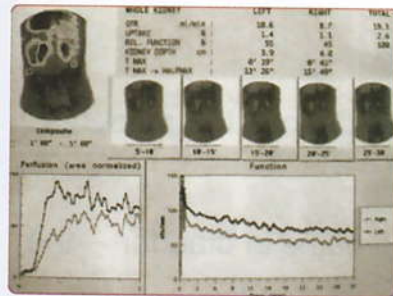
iii) Excretory phase where it leaves the kidney through the ureters.

-Indication: diagnosis of renal artery stenosis & obstructive uropathy

b) DMSA (dimercaptosuccinic acid)

- Idea: isotope is concentrated in renal tubules as only 5% is excreted → static kidneys images obtained 3 hours after injecting

- Indication: demonstration of renal scarring that result from chronic pyelonephritis, Focal defects as tumours & hematomas, lacerations, and ischemia



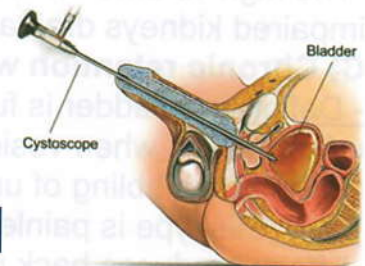
- Both types of radioisotope scanning are attached (labeled) with technetium^{99m} (^{99m}Tc) and both are given intravenous
- In obstructive uropathy administration of furosemide after isotope → accentuates abnormality in third phase of the curve

Endoscopy

-Advantage: great accuracy visualization of the urinary tract

- Uses: both diagnostic (urethroscopy, cystoscopy, ureteroscopy, and nephroscopy) and therapeutic purposes.

- Any suspicious lesion is biopsied



Urodynamic studies

-Advantages: study the physiologic functions of the bladder, urinary sphincters and urethra.

-Indications: for patients with neurogenic bladder & incontinence

- Include:

a) **Cystometry** → measures bladder volume against intravesical pressure

b) **Flowmetry** → measure the amount of urine passed per time unit.

- It is the most important, non-invasive and useful urodynamic test.

- It is commonly used to diagnose lower urinary tract obstruction as senile prostatic enlargement, urethral stricture & neurogenic bladder

The normal urine flow rate should be > 20 ml/second, provided a good amount of urine is voided (not <120 ml)

Retention of urine

Definition

Inability to pass urine in spite of a full bladder

Etiology

A) Mechanical causes

- 1- Urethral: Injury, phimosis, urethritis, stricture and stone.
- 2- Prostatic: Prostatitis, abscess, senile enlargement & cancer
- 3- Bladder: Bladder neck obstruction, stone and cancer.
- 4- Compression from outside by a pregnant uterus or pelvic tumours.

B) Functional (neurogenic) causes

- 1- Spasmodic → in post-operative cases, particularly after anal surgery (**acute retention**)
- 2- Atonic → in diabetic autonomic neuropathy & cauda equina

Types

A) Acute retention

- Definition: acute painful inability to pass urine despite the presence of the desire to void.

B) Chronic retention.

- Definition: patient voids but residual urine remains in bladder → the amount gradually increases → full bladder.

- As bladder distension is gradual → only little discomfort with hesitancy and reduction of force of stream.

- The high volume of retained urine → high intravesical pressure & impaired kidneys drainage

C) Chronic retention with overflow

- Definition: bladder is full with a large amount of residual urine & urine passes when vesical pressure exceeds urethral pressure

- Constant dribbling of urine occurs (**paradoxical incontinence**)

- C/P: this type is painless and the bladder is not tender

- Effect: produces back pressure on the upper urinary tract

Treatment

A) Acute retention

- The following measures are done in sequence:

1. Conservative treatment

- a) An analgesic is administered.
- b) Get out of bed and a warm bath is advised.
- c) If no organic obstruction (as postoperative retention) → injection of a parasympathomimetic drug as carbacol or doryl

2. Catheterization

- Indication: failure of conservative treatment

- Catheter used: A soft catheter (**Nelaton or Foley**)

- It may be removed or left till treatment of cause

- The commonest causes in males → senile prostatic enlargement & postoperative retention

- Retention is much less frequent in females

- Residual urine in SPE collects in the postprostatic Pouch

- **Differential diagnosis**

Anuria

- Definition: absence of urine or urine < 200mL/24h

- The bladder is empty (in contrary to retention where bladder is full & forms a median pyriform swelling in Suprapubic area → dull to percussion)

3. Percutaneous suprapubic catheter (**cystocath**)

- Indication: failure of catheterization

- Technique: under local anesthesia, puncture urinary bladder through abdominal wall in suprapubic region then introduce catheter

4. **Treatment of the cause** as prostatectomy

B) **Chronic retention**

1. **Treatment of the cause.**

2. **Catheterization**

- Catheter used: Self retaining Foley catheter

- Indications: back pressure on kidneys or elevated renal functions

- Slowly evacuation to avoid bleeding from either prostatic veins or renal decompression bleeding.

- The cause is investigated and treated later.

If there are no backpressure effects on the kidneys & renal functions are normal → no need for catheterization

Urinary incontinence

Definition

Uncontrolled escape of urine

Types

1- **Total incontinence**

- Definition: urine loss without warning

- Etiology: 2^{ry} to defective urethral sphincters as ectopia vesicae & sphincter injury after prostatectomy.

2- **Stress incontinence**

- Definition: urine loss in association with physical strain, e.g., coughing, laughing and straining.

- Incidence: common in multiparous women with weak muscle support of the bladder neck and urethra.

3- **Urge incontinence**

- Definition: involuntary loss of urine present with desire of micturition.

- Cause: acute cystitis & in UMNL

4- **Overflow (false) incontinence**

- Definition: involuntary dribbling of urine due to chronic retention or 2^{ry} to a flaccid bladder.

- Occurs when intravesical pressure exceeds urethral resistance

5- **Enuresis**

- Definition: bedwetting at night.

- This is a physiologic phenomenon during first **2-3 years** of life.

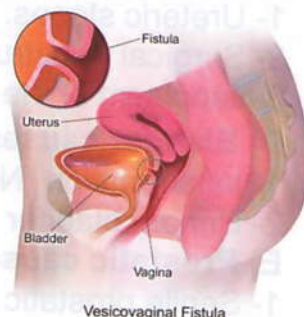
- If persists → may be: a) functional 2^{ry} to delayed neuromuscular maturation of urethrovesical component

b) A symptom of organic disease as neurogenic bladder.

6- **Fistula**

- Definition: abnormal communication between urinary & gynecological organs like vesico-vaginal and uretro-vaginal fistulas

- Etiology: follows obstetric trauma.



Hematuria

Definition

Passage of blood in urine

Types

A) Frank or microscopic (> 3 RBCs/HPF on urine analysis)

B) Painful or painless

C) In relation to urine stream

1-Total hematuria

-Definition→ passage of blood all through the stream

-Source →from the kidney or from massive vesical bleeding.

- Common causes → Stones, SPE & tumours

2-Terminal hematuria

-Definition→ passage of blood at the end of micturition.

- Source → pathology in trigone, bladder neck & sometimes senile prostatic enlargement (prostatic piles).

-Common cause → Shistosomiasis

3- Initial hematuria

-Definition→ passage of blood at the beginning of the act.

- Source→ urethra

Etiology

A) General causes 1- Anticoagulant therapy

2- Haemorrhagic blood diseases

as thrombocytopenic purpura or hemophilia.

B) Renal causes

1- Renal stones.

2- Congenital causes as polycystic kidney.

3- Inflammatory diseases as acute glomerulonephritis & TB

4- Traumatic (accidental, postoperative or post-instrumental)

5- Neoplasms of the kidney (Hypernephroma, transitional cell carcinoma and Wilm's tumour)

6- Vascular causes as renal infarction

C) Ureteric causes

1- Ureteric stones.

2- Surgical & instrumental procedures on the ureter.

3-Neoplasm of the ureter →transitional cell carcinoma

D) Bladder causes

1- Inflammation (Non-specific, Bilharzial or tuberculous cystitis)

2- Urinary bladder stones.

3- Neoplasms of the bladder.

E) Prostatic causes

1- Senile prostatic enlargement.

2- Prostate cancer.

F) Urethral causes

1- Urethritis

2- Urethral injuries

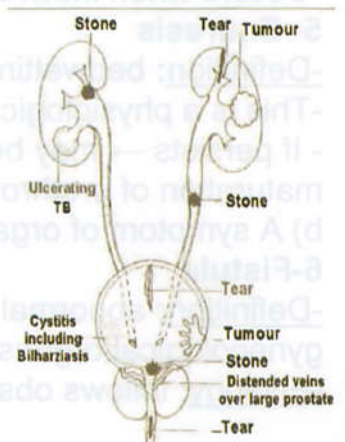
3- Urethral stones

4- Neoplasms of urethra

- Painless hematuria without other symptoms (silent) → regarded a symptom of tumour until proved otherwise (usually intermittent)
-Hematuria from kidney is usually associated with cylindrical clots
-Hematuria from bladder is associated with discoid clots.

- Common causes of hematuria

1. Urinary Stones (**most common**)
2. UT injuries
3. SPE
4. Renal tumours.
5. Bladder tumors
6. Shistosomiasis



Differential diagnosis

Other causes of red colored urine are:

1- Dietary

- a) Red beetroots
- b) Coloring agents in cakes, cold drinks & fruit juices.

2- Drugs, as

- a) Furadantin
- b) Rifampicin
- c) Laxatives that contain phenolphthalein.

3- Hemoglobinuria

-Definition: passage of haemoglobin in urine.

-Etiology: It occurs with haemolytic states

Evaluation

1-The cornerstone is a thorough:

- a) Medical history
- b) Directed physical examination
- c) Urine analysis.

2-For low-risk patients with microscopic hematuria

Renal ultrasonography and urine cytology

3-For patients with gross hematuria or risk factors

- a) Contrast-enhanced imaging of the kidneys & ureters (IVP or CT)
- b) Cystourethroscopy
- c) Urine cytology

Risk factors for significant urologic disease are:

- 1. Age > 40
- 2. Cigarette Smoking
- 3. History of exposure to possible carcinogens
- 4. Pelvic irradiation

Congenital anomalies of the urinary tract

Development of the Urinary Tract

A) Kidney, pelvicalyceal system and ureter

- 1- A ureteric bud arises from the lower end of the mesonephric (**Wolffian**) duct & grows upwards retroperitoneally.
- 2- This bud will form the ureter and its upper end will form the pelvis, calyces & collecting tubules (**excretory elements**).
- 3- The upper end of ureteric bud will be capped by metanephros → form glomeruli & nephric tubules (**secretory elements**).
- 4- Later, nephric tubules establish continuity with collecting tubules.

5- Ascent and rotation

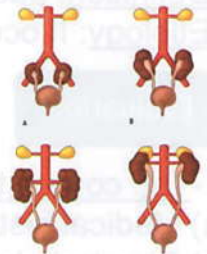
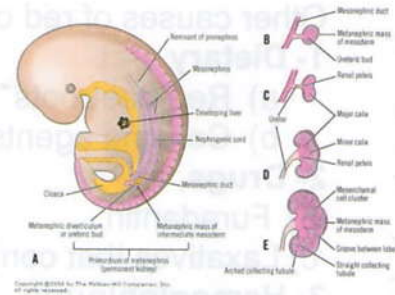
- Kidney starts development in pelvis then ascends to the loin.
- As the kidney ascends it also rotates
 - a) At first the pelvis lies anteriorly and the calyces posteriorly.
 - b) Later, pelvis rotates to lie medially while calyces face laterally

B) Urinary bladder

- Develops from the urogenital sinus (anterior division of cloaca), allantois and Wolffian ducts.

C) Urethra

- 1- The urogenital sinus forms:
 - i) The whole urethra in females
 - ii) Posterior urethra in males
- 2- The inner genital folds forms → anterior urethra in males
- 3- Tip of glans penis epithelium invagination → glandular urethra



Wolffian ducts form the trigone, ejaculatory ducts & vasa deferentia.

Anomalies of the kidney

Classification

Anomalies of ascent

- Ectopic (pelvic or iliac)

• Anomalies of rotation

- Incomplete rotation.

Anomalies of renal vasculature

- Aberrant & accessory renal arteries (vessels).
- Renal artery aneurysm and A-V fistula

Renal dysgenesis & cystic disease of the kidney

1. Hypoplastic
2. Simple renal (solitary) cyst
3. Polycystic kidney
4. Multicystic kidney
5. Medullary sponge kidney

Anomalies of number

- 1- Agensis:
Bilateral or unilateral.
- 2- Supernumerary

Anomalies of shape, form & fusion

1. Horse-shoe
2. Discoid (cake-shaped) kidney
3. Sigmoid & L-shaped kidney.

Anomalies of the renal pelvis

1. Bifid pelvis (double pelvis).
2. Pelvi-ureteric junction obstruction

Simple renal cysts

Pathology

- 1- May be solitary or multiple, unilateral or bilateral.
- 2- Mainly confined to the renal cortex and do not communicate with any part of the nephron or the renal pelvis

Clinical picture

1. Commonly asymptomatic
2. Renal mass (if cyst is huge)
3. Intermittent dull pain in flank or back.
4. Complications as hemorrhage or infection → acute symptoms

Differential diagnosis

- 1- Renal cell carcinoma
- 2- Renal abscess
- 3- Complex renal cysts (those which contain blood, pus, calcification, septation, irregular margins, or clusters of cysts) require further investigations (CT or MRI).

Treatment

1. For asymptomatic cases → no treatment but follow-up is required
2. If the cyst causes symptoms or hydronephrosis (due to renal or ureteric compression) treatment options include:
 - a) Percutaneous aspiration + injection of sclerosing agents or
 - b) Surgical excision of the cyst wall (**marsupialization**).
3. Complex cysts (haemorrhagic cyst, thick wall or cloudy fluid) → surgical exploration and excision.



Polycystic Kidney

Adult type (autosomal dominant)

Incidence

1. The common form of cystic disease of the kidney.
2. It affects both kidneys.
3. Responsible for **10%** of cases of end stage renal failure.
4. Almost **50%** have associated liver cysts (**liver function normal**)
5. Cysts of lung, pancreas and other organs may be found.
6. Intracranial aneurysms present in **30 to 40%** of patients

Pathology

1. Cysts do not form before the age of 10 years.
2. Grossly, kidneys are enormously enlarged with surfaces studded with cysts of various sizes
3. On section, the cysts are scattered throughout the parenchyma.
4. Cyst fluid usually amber clear but may be hemorrhagic or infected



Clinical picture

1- For the first 10 years of life

No symptoms (because kidneys are normal in function & anatomy)

2- From 10 to 30 years

Still asymptomatic but ultrasound show presence of cysts

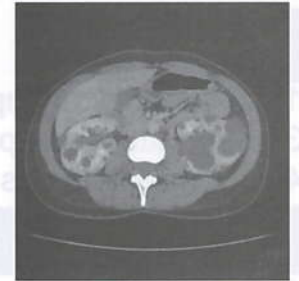
3- After 30 years, the patient may develop:

- | | |
|-----------------------------------|--------------------|
| a) Microscopic or gross hematuria | b) Hypertension |
| c) UTI | d) Flank pain |
| | e) Palpable kidney |

4- Between age 40 to 50.

-Renal function impairment (urea & creatinine start to elevate)

5- After the age of 50 → Renal failure



Investigations

A) Laboratory

1. Urine examination → RBCs or pus cells may be present
2. Blood urea and serum creatinine → elevated

B) Radiological

- 1- Ultrasonography or CT scan → accurate in diagnosis
- 2- Excretory urography with infusion shows enlarged renal shadows and bizarre calyceal pattern (**spider leg appearance**), as the calyces are stretched over the cysts

Treatment

A) Before renal failure

1. Control of hypertension by antihypertensive medications.
2. Treatment of UTI by appropriate antibiotics.
3. Low protein diet and regular check of renal functions
4. If a large cyst compresses the pelvis or upper ureter → by percutaneous ultrasound-guided aspiration.
5. Persistent heavy hematuria → embolization.

B) After renal failure

1. Renal transplantation (the definitive treatment)
1. Haemodialysis (temporary measure until a suitable donor found)

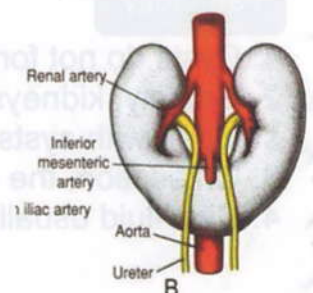
Horseshoe kidney

Pathology

- 1-Fusion occurs early in embryologic life (when kidneys lie in pelvis)
- 2- Ascent is arrested by the isthmus being blocked against IMA
- 3- Normal rotation not occur → renal pelvis lies on anterior surface of kidney → ureters ride over isthmus (which connects lower poles)
- 4- Angulation of the ureter as it crosses renal isthmus → ureteric obstruction → stasis → favours infection & stone formation

Incidence

More frequent in males



Clinical picture

- 1-One third of patients → asymptomatic
- 2- Symptoms of complications as pain, hematuria or fever.
- 3- A hydronephrotic horseshoe kidney → palpable below umbilicus

Investigation

- IVU** → a) Kidneys are lower in position
b) Lower poles are nearer to the midline than the upper poles
c) Lower pole calyces point medially and lie medial to the ureter
(**Flower vase appearance**)

Treatment

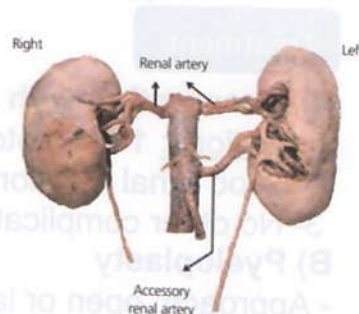
- Indication: only for complications
- Division of the kidney isthmus rarely required.



Undiagnosed hydronephrotic horseshoe kidney may excised by mistake in an exploratory laparotomy (very serious) → Before excising such a mass always check for the presence of the kidneys by U/S or by IVU

Aberrant and accessory renal vessels

- A single renal artery is noted in **75-85%** of individuals.
- Rarely, Aberrant arteries arise from vessels other than aorta or main renal artery.
- Accessory arteries → arise from aorta or main renal artery
- If one of these arteries passes to lower pole of kidney → may obstruct the ureter → hydronephrosis
- Division of an accessory artery → infarction of the corresponding portion of renal parenchyma (**end arteries**)
- On the contrary, aberrant renal vein can be divided without effects



Idiopathic pelvic-ureteric junction obstruction (PUJO)

Incidence

- A common cause of hydronephrosis in children & adolescents
- More often in males with the left side predominating.
- Bilateral obstruction occurs in at least **10 to 15%** of cases.

Pathology

- a) Intrinsic**: aberrant development of ureteric/renal pelvis circular smooth muscle → aperistaltic segment of the ureter at PUJO → obstruct urine outflow → hydronephrosis (progress or balanced)
- b) Extrinsic**: compression from aberrant or accessory vessel to the lower pole over which the PUJO runs

- Kidney is the commonest urinary organ to have anomalies
- Most renal anomalies are asymptomatic → need no treatment
- The anomalous kidney is liable to any disease that affects a normal kidney
- Polycystic kidney is a common cause of renal failure.

Clinical picture

Varies according to age:

- 1- Prenatal: Ultrasonography can diagnose condition in fetus
- 2- In infants: an abdominal mass (the most frequent)
- 3- In children: episodic intermittent flank pain following fluid intake, Vomiting may be present.
- 4- In adults: loin pain, recurrent UTI and hypertension

Investigations

1- Ultrasonography

- Diagnose condition exclusively in the prenatal period because of the wide use of maternal US
- Should be repeated postnatally after the first few weeks of life.
- It shows renal pelvis dilatation in the absence of a dilated ureter

2- Excretory urography → dilated pelvicalyceal system with abrupt contrast arrest at the PUJ

- The ureter is either nonvisualized or of normal caliber.

3- Micturating cystourethrogram (MCUG)

Is mandatory to rule out reflux

Treatment

A) Observation with serial US and renogram

- Indications:
- 1- asymptomatic stable cases
 - 2- Good renal functions
 - 3- No other complications (such as persistent infection or stones).

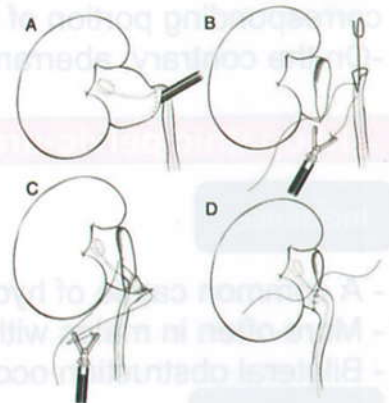
B) Pyeloplasty

- Approach: open or laparoscopic

- Indications:

- 1- Symptomatic children
 - 2- Significant hydronehrosis
 - 3- Impaired renal function (differential renal function $<40\%$ or prolonged drainage)
- Method: excision of the redundant part of the pelvis and proximal segment of the ureter and reconstruction of the pelvi ureteric junction

PUJO & posterior urethral valves are common anomalies that require early treatment to preserve renal function



Anomalies of the bladder

BLADDER EXTROPHY
(ECTPIA VESICAE)

PERSISTANT
URACUS →
UMBILICAL
FISTULA

CONTRACTURE OF THE
BLADDER NECK

VESICoureteric
REFLUX

Bladder exstrophy (ectopia vesicae)

Clinical picture

- 1- Lower part of anterior abdominal wall & anterior wall of the bladder are deficient.
- 2- Inner surface of posterior wall of the bladder bulges through the defect with its mucosal edges fused with the surrounding skin.
- 3- Urine flows onto abdominal wall from exposed ureteral orifices.
- 4- Bladder mucosa appears normal at birth, but persistent irritation and recurrent infections → chronic inflammation, metaplastic changes and sometimes malignant transformation.
- 5- Widening of symphysis pubis (connected by an intersymphyseal band) → characteristic waddling gait when the child begins to walk.
- 6- An umbilical hernia is always present (oblique inguinal hernias may be present as well)
- 7- Distorted pelvic anatomy → varying degrees of anal incontinence and rectal prolapse.
- 8- In males: a) associated epispadias with a short widened penis. B) Testes frequently appear undescended (are actually retractile)
- 9- In females: clitoris is cleft & associated vaginal anomalies.

Complications

1. Recurrent UTI
2. Sterility
3. Excoriation of the skin of the anterior abdominal wall.
4. Development of bladder cancer due to metaplastic changes.

Treatment

1-At birth, minimize trauma to the bladder mucosa by covering the bladder with plastic film & irrigate regularly with sterile saline

2- Functional reconstruction

-Timing: preferably in the first 48 hours of life

-Advantages:

- a) minimize bladder damage 2^{ry} to environmental exposure
- b) May avoid the need for Osteotomies

-Procedure: either one-stage repair or a 3-stage

i- Newborn:

- Closure of bladder, abdominal wall and posterior urethra.
- If > 72 hours → pelvic osteotomy (cutting bone to correct deformity) with external fixation.

ii- 6-12 month → epispadias repair.

iii- Later on, bladder augmentation or urinary diversion is required as bladder capacity is too small,
iv- when there is adequate bladder capacity & children can participate in voiding protocols → Bladder neck reconstruction and anti-reflux surgery is performed

Incidence

1 in 50,000 live births

- More common in males (4:1)

Embryological basis

Failure of cloacal membrane to be reinforced by ingrowth of mesoderm → ventral defect of urogenital sinus and overlying skeletal system



Vesicoureteric reflux (VUR)

Definition

Abnormal retrograde urine flow from bladder into upper urinary tract

Pathogenesis

-Reflux is normally prevented by:

- a) Low bladder pressures
- b) efficient ureteric peristalsis
- c) Ability of vesicoureteric junction to occlude the distal ureter during bladder contraction

- Reflux occurs when the normal antireflux mechanism is disturbed, especially when the intramural length of the ureter is too short.

- Etiology:

- a) 1^{ry} (due to congenital abnormality of the vesicoureteric junction)
- b) 2^{ry} (due to bladder neck obstruction)

Clinical picture

1- May be symptomless → discovered accidentally during micturating cystourethrogram (MCUG), IVU or renal ultrasound done for some other cause (**shows ureteric and renal pelvis dilatation**).

2- Symptoms of UTI: fever, dysuria, vomiting and diarrhea

3- Failure to thrive.

4- Loin pain (with a full bladder or immediately after micturition)

Investigations

A) Laboratory

Urine analysis and culture → UTI

B) Radiological

1- Ultrasound scan

2- MCUG (**the definitive test**)

- Diagnose and grade reflux and establish reversible causes

3- Urodynamicis → if suspicious of voiding dysfunction.

4- DMSA → to detect and monitor associated renal cortical scarring.

Management

1- Correct secondary causes

2- The majority of mild primary VUR will resolve spontaneously → initially observe with medical treatment:

- a) Timed voiding and double voiding
- b) Low-dose antibiotic prophylaxis → keep the urine sterile.
- c) Follow-up by renogram, US and MCUG.

Surgery:

- Indication: severe reflux or failure of medical treatment

- Approach: Laparoscopic or open ureteric re-implantation

Complications

- 1- Recurrent UTI and pyelonephritic scarring.
- 2- Reflux nephropathy → dilated calyces, renal parenchymal thinning and scarring
- 3- Hypertension
- 4- Progressive renal failure



Anomalies of the Urethra

Hypospadias

Definition

Hypospadias means that the urethral meatus opens on the ventral aspect of the penis at any point from glans penis to the perineum.

Pathology

- Incomplete development of the terminal part of urethra & corpus spongiosum.
- Missing urethral distal part is replaced by a fibrous band (**chordee**)

Etiology

1. Hormonal causes.
 - Deficiency of androgens or 5-alpha-dehydrotestosterone reductase enzyme during intrauterine life.
2. Deficient receptors in target cells

Classification

1. Glanular → Meatus is at the glans.
2. Coronal → at the coronal sulcus
3. Penile → at ventral surface of penile shaft (anterior, mid or posterior penile).
4. Penoscrotal → at the junction of penis and scrotum.
5. Perineal → Scrotum is usually bifid & urethra opens between its two halves

Clinical picture

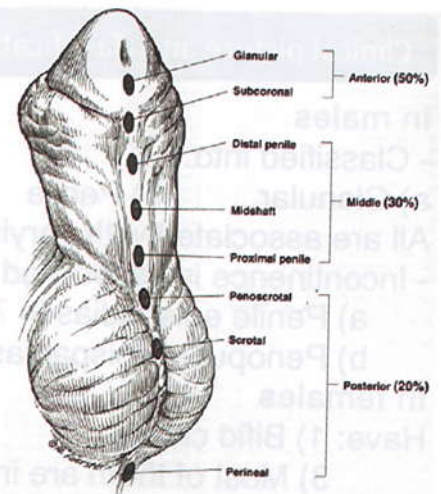
- 1- Meatus is ventrally placed and stenosed.
- 2- Prepuce is deficient venterolaterally.
- 3- Shaft is ventrally curved (due to the presence of chordee) except in the glanular variety.
- 4- Scrotum is bifid in the perineal type.
- 5- Associated lesions:
 - a) **10%** of patients have inguinal hernia or undescended testes.
 - b) **8%** of patients have upper urinary tract anomalies.
 - c) Patients with posterior hypospadias → have problem with sex differentiation

Investigation

Ultrasound → detect upper urinary tract problems

Incidence

1 in every 300 male children
 - Positive family history in 8% of cases
 - Hypospadias is anterior in **65%** of cases, mid penile in **15%** & penoscrotal or perineal in **20%** of cases



Treatment

- **Don't do circumcision because prepuce skin used for repair**

- **Timing:** at the age of **one year**.

- **Aim:** have a normally functioning male organ with normally situated meatus at tip of the glans.

- **The principles:**

1- Release the chordee → ventral curvature of the penis is corrected.

2- A new urethra is fashioned using neighbouring skin from the prepuce or the penile skin.

Surgery can be done in one stage (most cases) or two-stages (in difficult severe cases)

Epispadias

Definition

The urethral meatus opens on the dorsum of the penis (the reverse of hypospadias)

Incidence

Complete epispadias: 1 in 120,000 males and 1 in 450,000 **females**

Clinical picture and classification

In males

- Classified into:

a) Glanular b) Penile c) Penopubic d) Complete types

All are associated with varying degrees of dorsal chordee.

- Incontinence is associated with:

a) Penile epispadias in **75 %** of cases

b) Penopubic epispadias in **95%** of cases

In females

Have: 1) Bifid clitoris

2) Separation of labia

3) Most of them are incontinent

Treatment

- **Aim:**

1) To straighten the penis

2) Extend urethra to glans penis

3) Achieve continence, as patient grows → urethra lengthens and prostate enlarges → improve continence

- Glanular epispadias is not associated with incontinence
- Epispadias is a mild form of bladder exstrophy (both coexist in severe cases)

INJURIES OF THE URINARY TRACT

Renal Injuries

Incidence

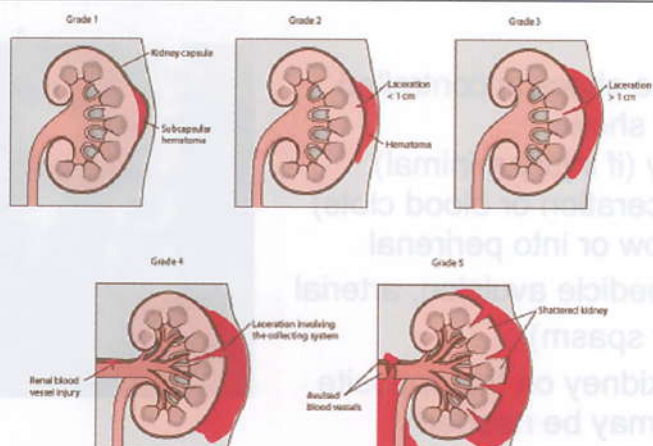
- Relatively rare due to:
 - 1- Kidneys are protected by rib cage & by heavy back muscles
 - 2- The kidneys are mobile in their adipose fat → flee away from the force of trauma.
 - 3- The fibrous capsule protects the parenchyma from splitting.

Etiology

1. **Blunt injuries** (the commonest **80-85%**) due to:
 - a. Direct trauma to abdomen (common), flank or back as in RTA
 - b. Indirect (less common) or contre coup injury, e.g., falling from a height and landing on the feet or buttocks.
2. **Penetrating wounds** e.g. knives or bullets (rare)
Associated visceral injuries are present in **80%**
3. **Iatrogenic injuries** occur during surgery

Grades of injury

1	Construction or non-expanding subcapsular haematoma No laceration
2	Non expanding perirenal hematoma Cortical laceration <1cm deep without extravasation
3	Cortical laceration >1cm deep without urinary extravasation
4	<u>Laceration</u> : through corticomedullary junction into collecting system Or <u>Vascular</u> : segmental renal artery or vein injury with contained haematoma
5	<u>Laceration</u> : shattered kidney Or <u>Vascular</u> : renal pedicle injury or avulsion



• Renal injuries are serious & may be complicated by injuries of other organs

Complications

1. Urine & blood extravasation → fibrosis around kidney → ureteric obstruction → hydronephrosis
2. Rupture, thrombosis or fibrosis → impair renal blood supply → atrophy or fibrosis
3. Renal ischemia → renovascular hypertension.
4. If haematoma becomes infected or liquified → a localized collection of clear amber fluid → Perirenal cyst.
5. Penetrating injuries → A-V fistula
6. Pseudohydronephrosis (Accumulation of urine all around kidney)

Clinical picture

Symptoms

1. Pain in the renal angle
2. Hematuria (Its degree is not proportionate to severity of injury)
 - Usually noted with the first voiding, but may be late to appear due to hypotension.
 - It may be continuous or intermittent.
 - About 30% of vascular injuries are not associated with hematuria.
 - Absent in: a) Complete pedicle avulsion b) Ureteric injury
3. Nausea, vomiting and abdominal distension due to intestinal atony (common in retroperitoneal bleeding)
4. Retention of urine (due to clots in the bladder)

Signs

General: Hypovolaemia in patients with severe renal injury

Local: 1- local tenderness or rigidity.

2- A mass in the flank (due to extravasated blood and urine)

3- Diffuse abdominal tenderness, abdominal distension and hypoperistalsis (if overlying peritoneum torn & leakage occur into the peritoneal cavity)

Investigations

A) Laboratory

1. Urine analysis (RBCs present in almost all cases)
2. Haematocrit value (Progressive anaemia = progressive Hge)
3. Renal function tests may be impaired from prolonged hypotension, bilateral injuries or injury of a solitary kidney.

B) Radiology

1-Ultrasound

- Show size & contour of kidney
- Visualize a collection around it.

2- Plain UT (X-ray film)

- Not show the injury but may show indirect evidence, e.g., obliteration of psoas shadow by a haematoma.
- Shows associated rib or spine fractures.

3- I.V.U.

- Visualize upper urinary tract as soon as the shock is controlled and blood pressure is $> 100 \text{ mm Hg}$. It may show:
 - a) Normal function & configuration of kidney (if injury minimal)
 - b) Deformed pelvis or calyces (if there is laceration or blood clots)
 - c) Contrast extravasation within renal shadow or into perirenal space
 - d) Non-visualization of kidney (total pedicle avulsion, arterial thrombosis or severe contusion $\rightarrow$ vascular spasm)
 - e) Confirms the presence of a functioning kidney on the opposite side (as nephrectomy of the injured kidney may be needed)



4-CT abdomen and pelvis with IV contrast (the most accurate)

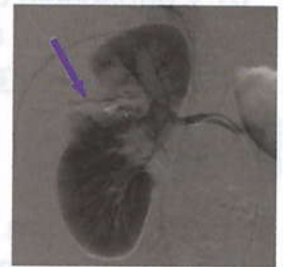
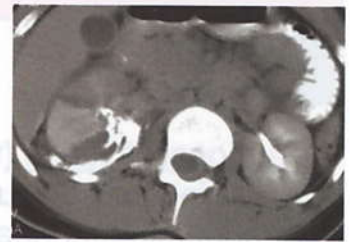
- Diagnose and grade renal trauma.
- It shows parenchymal lacerations, urinary extravasation, perirenal haematoma, non-viable renal tissue and other organ injuries

5- Angiography

- **Indication:** cases suspected to have renal artery thrombosis in order to place a stent across the thrombus.

6-Renal isotope scanning (DMSA)

- Useful in patients allergic to contrast material)
- Lack of uptake suggests injury or laceration of the renal artery.
- Areas of decreased activity compatible with renal contusion, while absence of uptake in one pole suggests amputation.
- Perirenal extravasation of radioactive urine may appear.



Treatment

A) Conservative

1- Hospitalization with bed rest

- Until hematuria has ceased & local signs of injury have subsided.

2- Analgesics for pain.

3-Large fluid intake

- To guard against clot retention and for hypovolaemia.

4- Broad spectrum antibiotics (guard against 2^{ry} infection)

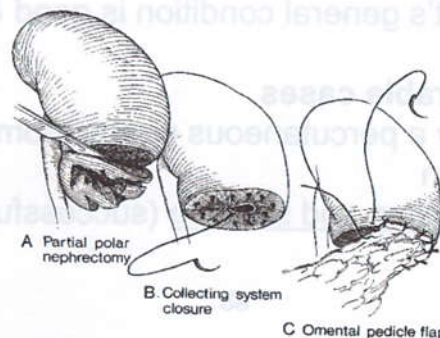
5- Follow-up parameters:

- a) Pulse, blood pressure & size of any perirenal mass.
- b) Repeated urine samples for gross & microscopic hematuria
- c) Haemoglobin and haematocrit estimations.

B) Surgical

Principles

1. Transperitoneal approach.
2. The colon is reflected medially.
3. Preliminary control of the vascular pedicle using vascular clamps.
4. A haematoma around the injured kidney is evacuated.
5. Debride traumatized and devascularized renal tissues
6. Small defects of cortical tissue are approximated by sutures.
- Large defects → fill by omental/ perirenal fat → obliterate dead space
7. Water-tight closure of the pelvicalyceal system.
8. Partial nephrectomy (if one pole of the kidney is avulsed)
9. Nephrectomy (if the kidney is shattered or with complete avulsion of vascular pedicle, provided that other kidney is functioning well)



Indications of surgical repair:

1. Persistent progressive hematuria or failure of vital stabilization
2. Presence of a progressive perirenal mass.
3. Evidence of perirenal infection
4. Penetrating injuries.
5. Renal pedicle injury (5%).
6. Associated intraperitoneal injury.
7. Indications for delayed surgery
 - a. hydronephrosis → relief of obstruction
 - b. hypertension → vascular repair or nephrectomy

Ureteric Injuries

Etiology

1-iatrogenic injuries (the most frequent)

- a) During difficult surgery or extensive gynaecological operations or abdominoperineal resection of the rectum
- b) Endoscopic urological procedures, e.g., cystoscopic ureteric catheterization, ureteroscopy and nephroscopy

2. Blunt or penetrating trauma (rare) 3. Pelvic radiation therapy

Clinical picture

1. Extravasation of urine which may be:

- a. Intraperitoneal → peritonitis or to urinary fistula (if there is drain)
- b. Extraperitoneal → enlarging urine collection at the site of injury
(A retroperitoneal collection → reflex paralytic ileus)

2. Ureteric obstruction, e.g., by a ligature.

- a) Complete obstruction → suppress urine production from corresponding kidney
- b) Partial obstruction → hydronephrosis

Investigations

1. Ultrasound: ureteric obstruction → pelvicalyceal system distension

2. Intravenous pyelography

- Shows distension of pelvicalyceal system, delayed secretion or extravasation of the contrast.

3. Ureteric catheterization and retrograde pyelography

- Will reveal obstruction and extravasation at the site of injury.

Treatment

Mainly Surgical

- Depends upon: a) the general condition of the patient

- b) Extent & site of injury c) Time of presentation

- The principles: a) Debridement b) Tension-free anastomosis

- c) Water-tight closure d) Ureteral stenting & drainage

- Operation:

1. **Early diagnosis (within the first week)**

- Exploration of the ureter & repair of injury by end-to-end anastomosis or by reimplantation of lower end of ureter into bladder.
(Provided that patient's general condition is good & surgeon is experienced)

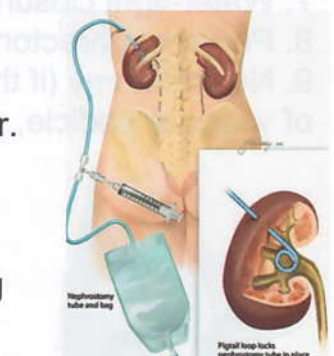
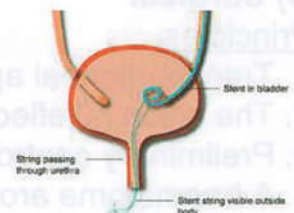
2. **Delayed unfavourable cases**

- Urinary diversion by a percutaneous nephrostomy and postponing ureteric reconstruction

3. Ureteric catheterization and stenting (successful in some cases)

- Preparatory ureteric catheterization
→ expose the ureter early in the procedure
→ avoid injury

- In patients with bilateral ureteric obstruction → anuria and acute renal failure



Urinary Bladder Injuries

	Extraperitoneal rupture (80%)	Intraperitoneal rupture (20%)
Etiology	1- Pelvic fracture (the commonest) 2. Open injuries (by stabs or bullets) 3. Surgical operations as hernia repair where there may be a sliding bladder, hysterectomy or abdomino-perineal resection. 4. During cystoscopic procedures as transurethral resection of the prostate or diathermy fulguration of a bladder tumour.	1. Blow or a kick to the lower abdomen, in a full bladder (the commonest)
Clinical picture	Symptoms 1. History of trauma → pelvic fracture 2. Intense desire to void. (as urine starts to collect in the retropubic space) but patient pass only few drops of blood-stained urine or none at all Signs 1. Hypovolaemic shock 2. A boggy swelling in the suprapubic area and rises upwards as urine extravasates between peritoneum & transversalis fascia (similar to intrapelvic rupture of the urethra) 3. DRE → prostate in its normal position (to differentiate it from intrapelvic rupture of urethra) 4. A necrotizing phlegmon (due to chemical irritation of anterior abdominal wall in untreated cases)	Symptoms 1. Sudden agonizing pain in the suprapubic area, later → dull aching pain all over the abdomen. 2. Severe oliguria or anuria (urine collects in peritoneum) Signs 1. Shock. 2. Peritonism, later peritonitis with tenderness, rebound tenderness and guarding all over the abdomen. 3. If amount of urine is large → abdominal distension & shifting dullness 4. DRE → fullness in rectovesical pouch. 5. Passing a urinary catheter brings no urine
DD	Intrapelvic complete urethral rupture DRE → prostate migrates up & felt higher than normal	
Complications	1. Pelvic abscess (from infected haematoma or urine collection) 2. Delayed peritonitis. 3. Partial incontinence (if bladder neck is injured)	

1. Treatment of renal injuries is mainly conservative
2. Most ureteric injuries are iatrogenic following surgical & endoscopic procedures
3. Bilateral ureteric ligation → anuria
4. The best way to avoid intra-operative ureteric injuries is to do preliminary exposure of the ureters
5. Bladder or urethral injuries should be suspected in patients with pelvic fractures
6. Extra pelvic injury of the urethra is diagnosed by triad of: retention of urine, few drops of blood at external meatus & perineal swelling

Investigations	1. <u>Plain X-ray</u> → pelvic fracture. 2. <u>I.V.U</u> → excludes other injuries & lack of UB filling 3. <u>Ascending cystogram</u> (give definite diagnosis) shows leakage of contrast outside the bladder
Treatment	1. Management of shock. 2. <u>Small extraperitoneal rupture with minimal extravasation</u> → Foley catheter drainage for 2 weeks till healing occurs. 3. <u>Large extraperitoneal rupture</u> - Need repair → bladder tear is exposed, its edges are trimmed and the defect is closed in two layers with polygalactin (vicryl). - A Foley catheter is left in bladder & a drain is placed in retropubic space. 4. <u>In intraperitoneal rupture</u> - Open the peritoneum → drain extravasated urine and to exclude any associated intraperitoneal injury 5. <u>Antibiotic prophylaxis.</u> 6. <u>Treatment of pelvic fracture</u> (according to its type). (internal fixation of the broken bone is contraindicated in the presence of urine extravasation, for fear of causing osteomyelitis)



Extraperitoneal



Intraperitoneal

- Bulbous urethral injury is more frequent than that of penile part
 - Membranous urethral injury is more frequent than that of the prostatic part
 - Catheterization is contraindicated in patients suspected of having rupture of the urethra → the proper treatment is to do suprapubic cystostomy

Urethral Injuries

Incidence

More common in men and are rare in women

Site

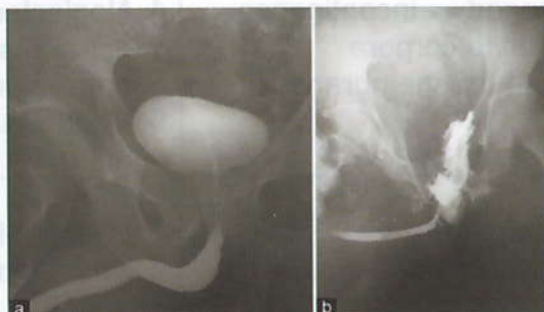
1. Anterior urethra (bulbous and penile).
2. Posterior urethra (prostatic and membranous).

Types

1. Complete or incomplete (according to circumference extent)
2. Total or partial (according to its depth in the wall)

	Injury of posterior urethra (membranous urethra = intrapelvic rupture)	Injury of Anterior (bulbous) Urethra (extrapelvic rupture)
Etiology	1. Severe trauma → pelvic fracture, e.g., RTA (similar to extraperitoneal rupture of the bladder, sometimes simultaneously injured) 2. Iatrogenic (by inexperienced transurethral instrumentation)	Perineal crushing forces: 1. Falling astride a hard object, e.g., bar of a bicycle (common) → crushing urethra against the pubis 2. A kick to perineum
Types	1. Mucosal tear & false passage (caused by faulty instrumentation) 2. Laceration of a part of the circumference. 3. Complete circumferential laceration. 4. Complete circumferential laceration with torn pubo-prostatic ligament → urinary bladder & prostate are thus free of any fixation → float upwards leaving a gap between the torn ends of urethra	1. Contusion. 2. Laceration that <u>does not involve</u> the whole Circumference) 3. Laceration that <u>involves</u> the whole urethral circumference
Complications	1. Blood loss & haemorrhagic shock 2. Deep extravasation of urine in the extraperitoneal space. 3. Injury of external sphincter (sphincter urethrae). If internal sphincter (Sphincter vesicae) is also damaged → incontinence 4. Impotence (if nerves to corpora cavernosa "adjacent to the membranous urethra" are injured) 5. Urethral stricture	1. Urine extravasation during voiding through superficial perineal pouch. 2. Stricture of urethra. 3. Urethral fistula. 4. Neglected cases get infected & sloughing of perineal skin may occur. 5. Periurethral abscess.
Clinical picture	Symptoms 1. History of severe trauma 2. Patient is unable to void urine though he feels the desire to micturate. Signs 1. Hypovolaemic shock. 2. Drops of blood at the external meatus 3. DRE → higher than normal floating prostate. 4. Distended urinary bladder (excludes extraperitoneal rupture of urinary bladder) • Catheterization is contraindicated in suspected injury → may compound the damage and introduce infection.	Symptoms 1. History of classic trauma 2. Perineal pain Signs 1. Passage of few drops of blood from anterior urethral meatus 2. Perineal haematoma. 3. Urine extravasation, if present, involves the perineum, penis, scrotum & lower part of anterior abdominal wall The triad of: 1. urethral haemorrhage 2. Perineal haematoma 3. Retention of urine is diagnostic

Investigations	1. <u>Ascending urethrogram</u> shows extravasation 2. <u>Plain x-ray</u> Assess fracture of pelvic bones 3. <u>Urgent intravenous urography (IVU)</u> Detect associated urinary tract injuries and to see the position of the urinary bladder. (DD: Extraperitoneal bladder rupture)	<u>Ascending urethrogram</u> shows the extravasation
Treatment	1. Blood transfusion & resuscitation 2. <u>A suprapubic cystostomy</u> In the emergency room, with no attempt at catheterization 3. The pelvic fracture is immobilized and the 4. Treatment of urethral stricture "if present" by either: a) Endoscopic visual urethrotomy or b) Surgery	1. If urethral injury is suspected, instruct patient not to try voiding. 2. Passage of catheter is contraindicated as it can: a) Transfer a partial tear into a complete one b) Introduces infection. 3. Immediate repair of the urethra is rarely required. A) Early treatment: 1. Contusion of bulbous urethra usually needs no treatment 2. If urethrogram shows extravasation of contrast Material → diversion of urine by suprapubic cystostomy & give prophylactic antibiotics 3. An expanding haematoma of perineum or infected urine extravasation → incisions & drainage B) Later treatment After <u>3 weeks</u> the urinary bladder is filled with a contrast material and a <u>micturating cystourethrogram</u> is obtained. 1. If urethra is normal → remove the suprapubic tube 2. If stricture develops → endoscopic urethrotomy followed by repeated dilatation 3. For tight & long strictures → surgical correction



Urinary tract infections

Non-specific Infections

Incidence

More frequent in females than males

Etiology & Pathology

Causative organisms

- 1- Escherichia coli (E. coli) in 80%
- 2- Proteus mirabilis
(Urea-splitting organism → ammonia & alkalinizes urine)
- 3- Pseudomonas aeruginosa (with urinary stasis).
- 4- Gram Positive Cocci (Staphylococci & Enterococci) → rare
- 5- Chlamydia
(With diabetes, prolonged antibiotic abuse & reduced immunity)

Route of infection

- 1- Ascending Infection (the most common)
Perineum organisms ascend through urethra (especially in females)
- 2- Haematogenous & lymphatic spread → rare.

Predisposing factors

- 1- Females (particularly the sexually active) because:
 - a) They have a short urethra.
 - b) During sexual intercourse, bacteria are transferred from the vulvo-vaginal and urethral areas into the bladder.
 - c) Pregnancy is associated with functional ureteric slow peristalsis → a higher incidence of pyelonephritis.
- 2- Defective immunity as DM & immunosuppression
- 3- Urinary stasis which may be:
 - a) Mechanical as stones, or
 - b) Functional as neurogenic bladder & vesico-ureteric reflux
- 4- Urethral instrumentation → push normally colonized bacteria

Types

1-Uncomplicated UTI

- No structural abnormality & infection is harmless to kidneys
- A short course of an antibiotic eradicates the infection.

2-Complicated UTI

- Accompanied by obstruction
- A combination of upper UTI and obstruction → rapid renal damage
- Respond slowly to antibiotics & needs relief of obstruction

Complications

- 1- Bacterial persistence & chronicity if not properly treated.
- 2- Septicaemia and septic shock.
- 3- Stone formation
- 4- Impairment of renal function → end in renal damage

Non-Specific UTI

Basic facts

- The main organism: E-coli from colon
- Females > males
- Main predisposing factors: urinary stasis & catheterization
- Uncomplicated UTI is easy to treat.
- UTI complicated by obstruction is difficult to treat & is so serious if obstruction is high.
- Treatment Includes:
 1. Antibiotics against Gram negative bacilli.
 2. Relief of obstruction
 3. High fluid intake.

Investigations

Laboratory

(Identify organism & its antibiotic sensitivity)

1- A clean catch mid stream urine (MSU) specimen

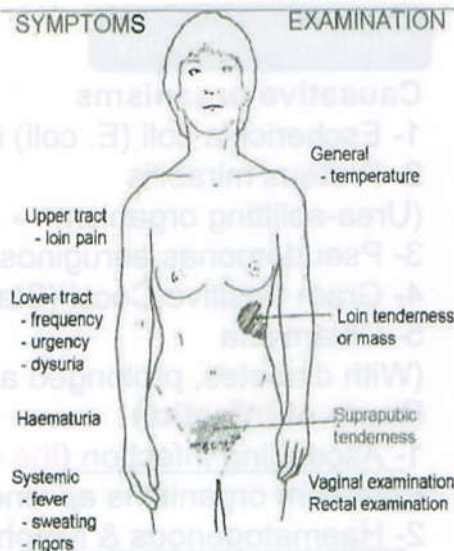
- Obtained & cultured immediately.
 - In babies → specially designed bag attached to genitalia
- 2- Pyuria (pus cells in urine)
- 3- Bacterial counts $> 10^5$ /ml

Radiological

(Exclude structural & pathological abnormalities)

- 1- Plain x-ray (KUB)
- 2- Ultrasound
- 3- IVU & urine flow rate → needed in selected cases:
 - a) Upper tract infections (e.g. pyelonephritis) specially if with fever
 - b) Infections in children (to look for congenital anomalies)
 - c) Persistent bacteriuria
 - d) Recurrent infections

Clinical picture



Treatment

- 1- High fluid intake → induce diuresis
- 2- Antibiotics (**mandatory for all cases**)
 - a) Route: (i) Oral → for most cases
(ii) IV → severe cases of pyelonephritis with fever & rigors
 - b) Choice: depends on culture & sensitivity; the most commonly used are: (i) Trimethoprim/sulphamethoxazole (septrin)
(ii) Nitrofurantoin (iii) Quinolones
- 3- Relief of obstruction (may be urgent)
- 4- Drainage of an abscess (if present)
- 5- Prophylaxis (for recurrent cases) by:
 - a) Fluid intake (at least **2 liters per day**)
 - b) Perineal hygiene especially in females.
 - c) Regular voiding.
 - d) Avoid constipation (which may impair bladder emptying)
 - e) Prophylactic small doses of antibiotics for a few months.

Pyelonephritis

Definition

Infection of the renal parenchyma and pelvis

Incidence

Commoner in females particularly during pregnancy

Etiology

Causative organism:

- 1- E. coli (the usual organism)
- 2- Proteus

Route of infection:

- 1- Ascending (usually)
- 2- Haematogenous (less commonly)

Complications

- 1- Septicemia and septic shock
 - 2- Pyonephrosis (Urine flow obstruction → dilatation → a bag of pus)
 - 3- Renal damage (due to combination of obstruction & infection)
 - 4- Perinephric abscess.
 - 5- Chronic pyelonephritis.
- Scarring of the kidney → renal hypertension.
- If bilateral → chronic renal failure

Clinical picture

Symptoms

1. Sudden severe loin pain
2. Fever & rigors
3. Vomiting
4. Frequency, burning micturition & dysuria

Signs

1. Temperature rises up to 39-40°C.
2. Tenderness in the renal angle & sometimes muscular rigidity.
3. Bilateral pyelonephritis → give features of uraemia.
4. In severe cases, ill looking patient if not treated → septic shock

Investigations

Laboratory

- 1- Mid-stream urine sample (MSU) → for culture & sensitivity
- 2- Blood urea and serum creatinine.
- 3- Blood sugar and electrolytes.

Radiological

Ultrasound → hydronephrosis is cases of obstruction

Treatment

A) Pyelonephritis in children

Vesico-ureteric reflux (VUR) is the usual cause of pyelonephritis in children

B) Non-obstructed acute pyelonephritis

- 1- Rest in bed
- 2- Adequate hydration by drinking a large amount of fluids.
- If there is vomiting → give IV fluids & antiemetics
- 3- Analgesics & antipyretics as NSAIDs
- 4- Alkalinization of urine → relief dysuria & inhibit organisms growth
- 5- Antibiotics → **for 3 weeks**
(Against Gram -ve bacilli until culture & sensitivity result is available).

It may be acute or chronic

- Distinction between non obstructed & obstructed acute pyelonephritis is important
- The classic picture of acute Pyelonephritis is acute onset of fever, chills, flank pain, pyuria & bacteriuria

- Differential diagnosis

1. Basal pneumonia and pleurisy.
2. Acute cholecystitis (DD by U/S)
3. Acute appendicitis. (The initial pain is periumbilical)
4. Acute pancreatitis.
5. Perinephric abscess.



C) Obstructed acute pyelonephritis

In addition to the measures of the non-obstructed cases:

Urgent drainage of the obstructed kidney (mandatory)

- By: 1- ultrasound-guided percutaneous nephrostomy or
2- Cystoscopic placement of a ureteric catheter
(Extends from renal pelvis to bladder)
- When the patient improves → deal with cause of obstruction

- Urological investigations are done to exclude underlying abnormality.
- Repeated urine cultures done to ensure cure

Pyonephrosis

Definition

Retention of infected urine and pus in the kidney

Etiology

- 1- Primary: due to both infection & obstruction as in renal calculi
- 2- Secondary: due to infection of a pre-existing hydronephrosis.

Complications

- 1- Septicemia & septic shock
- 2- Renal scarring and damage

Clinical picture

Symptoms → Loin pain

- Timing: worse at night
- Character: constant aching
- Renal colic with passage of pus or stones down the ureter

Signs

General: → **Fever**

- Evening rise of temperature to 37.5° or 38°C.
- Considerable rise may occur from retention of purulent urine.

Local:

1- **Swelling**.

- The kidney is palpably enlarged, tender and with limited mobility.
- Fluctuation cannot be elicited.
- Usually small but if due to infection of hydronephrosis → very large

2- **Pyuria** → cystitis

- Turbid Urine & contains much pus → settles to glass bottom

Pathology

- The pelvicalyceal system is transformed into multilocular cavity filled with pus & lined with necrotic renal parenchymal tissue.
- There is marked perinephritis with dense adhesions and fibrofatty proliferation
- 2^{ry} calculi with calculus pyonephrosis are common
- Differential diagnosis
Similar to that of acute Pyelonephritis

Investigations

Laboratory → Urine analysis and culture

Radiological

- 1- Abdominal ultrasound → shows dilated kidney with pus inside
- 2- A plain x-ray → may reveal a calculus.
- 3- CT scan → more accurate in detecting stones



Treatment (Urgently)

1- Antibiotics and kidney drainage by:

- Inserting a large percutaneous nephrostomy tube or
- Ureteric catheter.

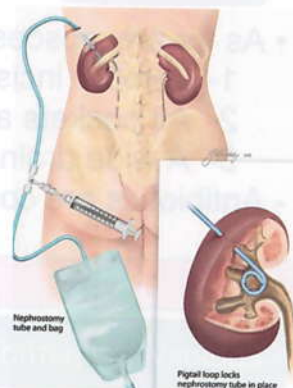
-Any cause of obstruction as a stone or stricture → corrected

2- Nephrectomy

-Indication → advanced cases with atrophic parenchyma and total renal function loss (as detected by radioisotope renal scan)

- Prerequisite → opposite kidney is normal

- Preliminary nephrostomy may done first



Perinephric abscess

Definition

Suppuration of perinephric fat and fascia

Etiology

1- Primary perinephric abscess (rare)

- Blood-borne from a distant septic focus as a boil or carbuncle

2- Secondary perinephric abscess (more frequent)

- Due to extension of infection from the kidney or a neighboring structure, e.g. spine, pleura or pelvic organs

Clinical picture

Symptoms → Loin pain

- Increases by: movement, respiration & coughing

Signs

General: Hectic fever

Local:

1- Loin fullness, tenderness and rigidity.

- Swelling is ill defined and does not move with respiration.

2- Edema & skin redness (if the abscess extends superficially)

3- Slight eversion & flexion of hip joint (from psoas muscle spasm)

Investigations

Laboratory

1- Urine analysis → usually sterile

2- CBC → polymorphonuclear leucocytosis

Radiological

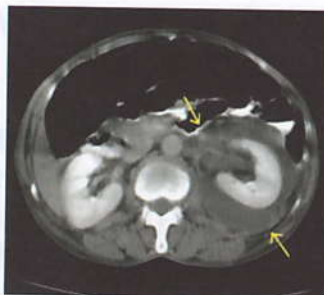
1- Ultrasound or CT scan (**diagnostic**) → pus around kidney

2- Plain X-ray show:

a) Scoliosis with the concavity towards the abscess side

b) Obliteration of the psoas shadow

c) Elevation and fixation of the diaphragm



Incidence

A perinephric abscess usually affects adults, more on RT side

Pathology

-The infecting organisms: staphylococci, less commonly → streptococci & E. coli.

- Pus collects between kidney and perinephric fascia → extend downwards into pelvis along course of the ureter or may burst through perinephric fascia → reach the loin.

- Complications of neglected abscess → septicaemia & septic shock

Treatment

- As for any abscess → **drainage of pus** → through
 - 1- Lumbar incision under general anaesthesia.
 - 2- All pockets are thoroughly opened.
 - 3- A wide drain is inserted.
- Antibiotics are complementary

Cystitis

Etiology and pathology

Causative organisms

- 1-E. coli (**the commonest**)
- 2- Others: Proteus, Strept. faecalis & Pseudomonas aeruginosa

Predisposing factors

- 1-Incomplete bladder emptying → accumulation of residual urine, e.g., prostatic obstruction, urethral stricture & neurogenic bladder
- 2- Lowered general resistance, e.g., diabetes.
- 3- Urinary instrumentation, calculi, F.B & bladder neoplasms

Routes of infection

- 1- Ascending (**commonest**)
 - From the urethra, prostate and vulva or during instrumentation
- 2- Descending from the kidney.
- 3- Direct through a vesicovaginal or vesicointestinal fistula

Clinical picture

Symptoms

- 1- Increased frequency (diurnal & nocturnal) → **main symptom**.
- 2- Suprapubic and perineal pain.
 - Timing: At the end of micturition
 - Referral: to the glans penis or labia majora

Signs

- 1- Pyrexia rarely in acute cases and it seldom reaches 38°C.
- 2- Tenderness
Over bladder on abdominal, rectal or vaginal examination
- 3- Pyuria → cloudy urine & feathery deposit may appear on standing
- 4- Hematuria
 - Terminal hematuria → the last few drops of urine are blood-stained
 - In severe cases → whole specimen may be blood-stained

Investigations

Laboratory → Examination of the urine

- Mid-stream specimen is sent for analysis and culture & sensitivity

Instrumental → Cystoscopy (**contraindicated in acute stage**)

In chronic cases → reveal the precise pathological lesions & local predisposing causes in bladder, urethra or prostate as bladder ulcer

Treatment

follows the previously mentioned general principles

Prostatitis

Acute prostatitis

Etiology

Causative organisms

- 1- Escherichia coli (**most common**)
- 2- Others: Staphylococcus aureus, Staphylococcus albus, Streptococcus faecalis, Neisseria gonorrhea or Chlamydia

Route of infection → from the posterior urethra.

Clinical picture

Symptoms

- **General:** (overshadow the local)
 - 1- The patient feels ill, shivers, aches all over (especially in the back)
 - 2- The temperature may be up to 39°C.
- **Local:**
 - 1- Pain on micturition
 - 2- Urine contains threads in the initial voided sample → cultured
 - 3- Perineal heaviness, rectal irritation & pain on defecation
 - 4- Frequency (when infection involves the bladder)

Signs

- DRE** → a) Tender prostate
b) One lobe may be swollen more & seminal vesicles involved
c) A frankly fluctuant abscess (uncommon)

Treatment

Prolonged treatment with an antibiotic that penetrates the prostate as → trimethoprim or fluoroquinolone

- May easily be diagnosed as Having influenza
- **Complications:**
Spread of infection to the epididymis & testes
- Treatment must be vigorous & prolonged to eradicate infection & avoid recurrent attacks.

Prostatic abscess

Clinical picture

Symptoms

- 1- Rising steeply temperature with rigors (masked by Antibiotics).
- 2- Severe, unremitting perineal and rectal pain with occasional tenesmus → confused with an anorectal abscess.
- 3- Urine retention → best treatment is suprapubic catheterization

Signs

- 1- **DRE** → prostate is enlarged, hot, extremely tender & fluctuant

Treatment

- 1- Drainage without delay by: a) Transurethral evacuation or
b) Transperineal aspiration and drainage.
- 2- Empiric antibiotic till results of pending culture with fluoroquinolone, e.g., ciprofloxacin.

Chronic prostatitis

Etiology

- Inadequately treated acute prostatitis
- **Causative organism** → Chlamydia

Clinical picture

Symptoms → of posterior urethritis, prostatic pain and perigenital pain accompanied by intermittent fever

Signs

- **DRE** → normal or may show a soft, boggy & tender prostate

Diagnosis

Laboratory

1- The three-glass urine test is valuable.

If 1st glass with the initial voided sample → urine containing prostatic threads → prostatitis is present

2- Prostatic fluid examination obtained after prostatic massage → shows pus cells & sometimes bacteria.

Instrumental → Urethroscopy → inflammation of the prostatic urethra & pus may be seen exuding from the prostatic ducts.

Treatment

Antibiotics for a long period of time, probably a few months

- Trimethoprim & fluoroquinolones → penetrate into prostate
- If Chlamydia is suspected → doxycycline (is of choice)

The diagnosis of chronic prostatitis is difficult, In many cases all investigations are normal

Urethritis

Gonococcal urethritis

Clinical picture

- The incubation period is **3-10 days**.
- May be asymptomatic.
- In males → urethral itching, urethral discharge and dysuria.
- Without treatment, urethritis will stay for **3-7 weeks**.
 - **95%** of men become asymptomatic after **3 months**.
- Spread of infection to: a) prostate → prostatitis
b) Epididymis → epididymo-orchitis.

Investigations

1- A gram-stained smear (the usual method)

- Positive if gram -ve diplococci are seen within polymorphonuclear leucocytes.

2- Alternative diagnostic methods are based on detection of: gonococcal enzymes, antigens, DNA, and liposaccharides.

- Gonorrhea is a sexually-transmitted disease.
- Neisseria gonorrhea are Gram-negative diplococci located within the neutrophils.
- Common site of infection → urethra
- Concurrent infections with Chlamydia & others are common.

Complications

- 1- Periurethritis → abscess formation → urethral fibrosis → urethral stricture
- 2- Prostatitis
- 3- Epididymitis
- 4- Tenosynovitis
- 5- Arthritis

Treatment

- 1- Antibiotic treatment.
- 2- Treatment of the sexual partner is mandatory
- 3- Sexual intercourse should be avoided until cure

Differential diagnosis

Non-gonococcal urethritis → discharge is scant & clear

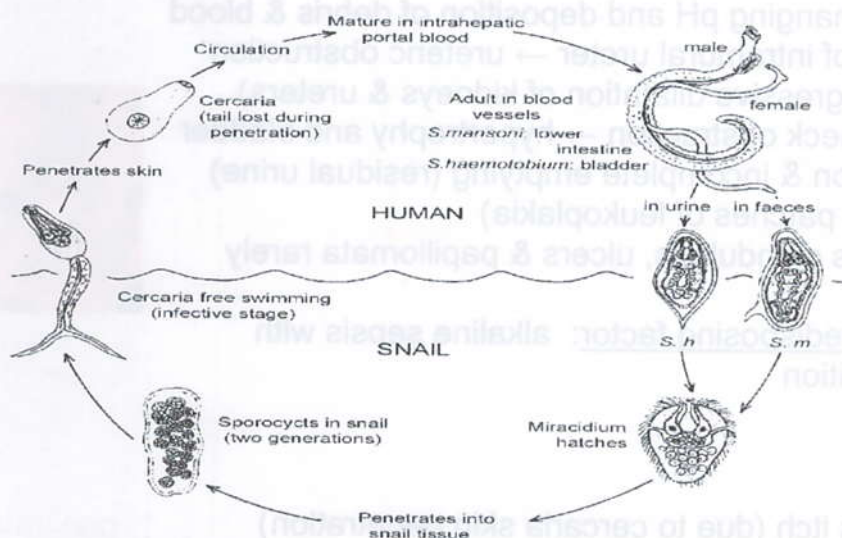
Specific infections

Urinary Bilharziasis (Schistosomiasis)

Incidence

- Affects adolescents and adults between ages of **10 and 30**.
- Males suffer more than females (4: 1).
- The majority has history of exposure to water canals especially of small streams, such as farmers and fishermen

Life cycle



Etiology

Causative organism

- 1- Schistosoma haematobium (96%)
 - 2- Schistosoma mansoni (4%)
- The worms migrate from the liver to the mesenteric veins → anastomotic channels between haemorrhoidal and vesicoprostatic plexuses → urinary bladder

Non-gonococcal urethritis

- Incidence: 50% of urethritis cases

Etiology:

Mainly → Chlamydia trachomatis

Clinical features

- Incubation period **7-21 day**

Symptoms:

1. Dysuria
2. Mild whitish or clear scanty urethral discharge (may be absent & the patient only complain of urethral itching)
- 3.

Asymptomatic (In contacts of women with known cervical chlamydial infection)

Treatment

Tetracycline (For both the patient & sexual partner)

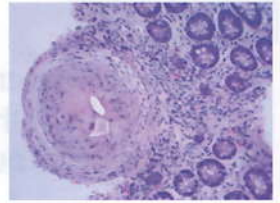
Pathology

Site

The urinary bladder (the commonest)

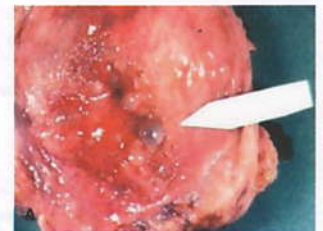
Gross picture

- 1- Patchy hyperaemia over the trigone.
- 2- Bilharzial **sandy patches** (aggregations of calcified dead ova in the Submucosa → appear through mucosa like sand under water).
- They form a barrier to the passage of ova → so normal urine does not mean a cure of the disease
- 3- Bilharzial tubercles.
- 4- Bilharzial nodules (bodies that may have a pseudoglandular or cystic appearance (**cystitis glandularis** and **cystitis cystica**)).
- 5- Bilharzial papillomata
- 6- Bilharzial granulomata (Bilharzial granulation tissue submucous masses → smooth tumour-like swellings projecting into bladder)
- 7- Bilharzial ulcers.



Complications

- 1) 2^{ry} bacterial infection (**common**) → acute or chronic cystitis.
- 2) Stasis → stones → changing pH and deposition of debris & blood
- 3) Infiltration & fibrosis of intramural ureter → ureteric obstruction (Usually bilateral → progressive dilatation of kidneys & ureters)
- 4) Fibrosis → Bladder-neck obstruction → hypertrophy and bladder trabeculation → dilatation & incomplete emptying (residual urine)
- 5) Malignancy (starts in patches of leukoplakia)
- Cystitis cystica, cystitis glandularis, ulcers & papillomata rarely Precancerous
- The most important predisposing factor: alkaline sepsis with ammoniacal decomposition



Clinical picture

Symptoms

- Swimmer's or bather's itch (due to cercaria skin penetration)
- **After 4-12 weeks**: fever, urticaria & asthma (may pass unnoticed)
- **Within 6 months** → ova invade the bladder leading to:
 - a) Hematuria (cardinal symptom) → slight (few drops) & terminal.
 - b) Pain
 - Character: urethral burning
 - increase in cases of: ulceration, stone formation or malignancy
 - c) Frequency
- Cause: trigone congestion but later follows 2^{ry} cystitis

Investigations

Laboratory

Urine examination

- In acute congestive stage → urine looks smoky & contains living bilharzial ova & RBCs
- When 2^{ry} infection occurs → becomes alkaline and turbid with blood, pus, phosphates, epithelial debris and dead ova.

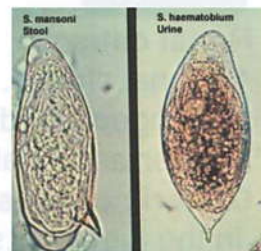
Radiological

1- Plain x-ray → Bilharzial calcification of bladder, terminal ureters, seminal vesicles (**honey comb pattern**), calcified papilloma or calculi.

2- IVU shows

Strictures of t ureters, hydronephrosis or bladder neck obstruction

Instrumental → Cystoscopy → Bilharzial lesions & take biopsy



Treatment

- 1- Anti-Bilharzial drugs.
- 2- If 2^{ry} cystitis is present → urinary antiseptics
- 3- Surgical treatment (depends on the individual lesion)
 - a) Cystitis cystica, cystitis glandularis & Bilharzial papillomata only if bulky and cause obstruction → Cystoscopic fulguration or excision biopsy
 - b) Superficial bladder ulcers → cystoscopic excision.
 - c) For severe cases & for deep ulcers → Partial cystectomy
 - d) Bladder neck obstruction → cystoscopic incision or resection
 - e) Lower ureteric stricture → ureteroscopic balloon dilatation or ureterovesical implantation



Obstructive uropathy

General principles

Etiology

Renal causes

- 1- Renal stones.
- 2- Congenital (idiopathic) pelvi-ureteric junction obstruction (PUJO)
- 3- Aberrant renal vessels.
- 4- Renal pelvic tumors.

Ureteric causes

- In the lumen:
1. stones
 2. Blood clots.
- In the wall:
1. Ureteric stricture
 2. Bilharzial
 3. TB
 4. Iatrogenic
 5. Tumors

Outside the lumen (compression)

1. Infiltration by carcinoma of cervix, colon, or rectum.
2. Idiopathic retroperitoneal fibrosis.

Urinary bladder causes

1. Tumours involving uretero-vesical junction.
2. Neurogenic bladder dysfunction

Bladder neck causes

1. Congenital bladder neck obstruction.
2. Bilharzial bladder neck obstruction.

Prostate causes

1. Benign prostatic hyperplasia (BPH).
2. Carcinoma of the prostate

Urethral causes

1. Congenital: meatal stenosis & posterior urethral valves.
2. Stricture of the urethra

Pathological changes

All occur above the obstruction

Urethra

Increased hydrostatic pressure → urethral dilatation

Urinary bladder

a) Early (in the compensated phase)

- Increased urethral resistance → bladder muscle layer hypertrophy
- The bladder wall becomes trabeculated.
- Mucosa protrudes between muscle fibres → **sacculles**
- When mucosa bulges outside bladder wall → **Diverticula**

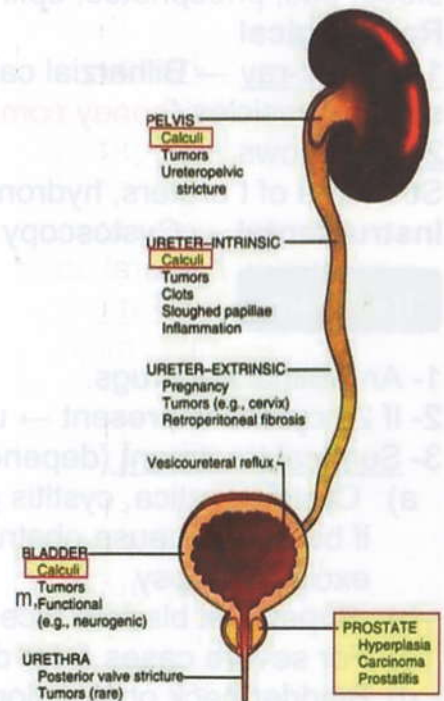
b) Later with unrelieved obstruction

- Bladder dilates & its wall thins out → atony → urine retention

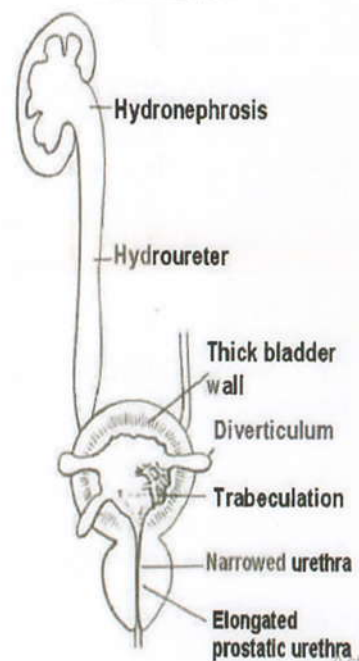
Ureter

a) Early → muscular hypertrophy of the wall.

b) Later → atony → hydroureter (dilated, elongated & tortuous)



Renal failure



Kidney

a) Morphological changes

- Pressure in pelvi-calyceal system increases → **hydronephrosis**
- The first to suffer are calyces → Concave (cupping)" normal "→ flat → rounded (clubbing) → ballooned
- The increasing intrapelvic pressure → compress renal parenchyma against renal capsule → ischaemic atrophy → thinning out
- Later → complete kidney destruction (thin-walled sac filled with clear fluid or pus if infected)

b) Functional changes

- Intra-pelvic pressure continues to rise till it overcomes the glomerular filtration pressure → urine formation stops.
- The contralateral normal kidney undergoes compensatory hypertrophy in order to maintain an overall normal renal function.
- In neglected bilateral obstruction → renal failure

c) With relief of obstruction

- Early treatment → major morphological & functional improvement
- Delayed treatment → some degree of irreversible renal damage

d) Factors affecting changes following obstruction

1- Level of obstruction.

- Obstructive lesion at or below bladder neck → bilateral effect
- Lesion in ureter or PUJ → affects one kidney only

2- Degree of obstruction.

Severe obstruction → more damage

3- Duration of obstruction.

- Long-standing partial / intermittent obstruction → marked dilatation
- Acute obstruction → mild dilatation but rapid functional deterioration

4- Development of infection on top → serious deterioration

Complications

- 1- Stasis → infection → further faster functional kidney deterioration
- 2- Stone formation.
- 3- Renal insufficiency & finally failure (in bilateral cases)

- Normally, the pressure within the renal pelvis is close to zero
- Obstructive urinary conditions that require urgent relief

1. Acute urine retention
2. Calcular anuria
3. Accidental ligation of both ureters at surgery
4. Infection on top of obstruction → accelerated functional deterioration & septicaemia

Hydronephrosis

Definition

Chronic aseptic dilatation of renal pelvis and calyces due to partial or intermittent obstruction of the urinary tract (unilateral or bilateral)

Etiology

- 1- All causes of chronic obstructive uropathy
- 2- Vesicoureteric reflux



- 3- Pregnancy (hydronephrosis especially on right side)
 - Temporary & subsides within **2- 12 weeks** after delivery
 - Onset: 1st few weeks & reaches maximum within 5th– 6th months
 - Cause: a) high progesterone level → ureteric musculature atony
b) Fetal head may press both ureters

Differential diagnosis of palpable hydronephrosis

- 1- Polycystic kidney.
- 2- Hypernephroma

Clinical picture

- 1- Pain
 - Character: slight dull aching or a sense of discomfort in the loin.
- 2- If large → Swelling (a cystic swelling that fills the renal angle)
- 3- If advanced & bilateral → manifestations of renal failure

Investigations

Laboratory

- Urine analysis → RBCs, crystals and pus cells

Radiological

1-Ultrasound examination

- Detect renal size, renal cortex thickness & the cause, e.g., stones.

2- IVU → will show the following

- a) Dilatation of renal pelvis → becomes biconvex.
- b) Pelvi-ureteric junction is distorted and is raised upwards.
- c) Loss of cupping & clubbing then ballooning of the minor calyces (The major calyces are also dilated)
- d) Delayed contrast excretion & medium concentration is below normal (In advanced cases no secretion occurs)

3- Ascending (retrograde) pyelography

- To confirm diagnosis and to see the cause



Treatment

- 1- Removing the cause of obstruction → recovery of the kidney.
- 2- Nephrectomy
 - Avoided as possible. However, it is justified in the following:
 - a) The kidney is non-functioning with infusion urography.
 - b) Sonography showed very thin parenchyma.
 - c) Renal scan shows zero function.
 - d) The opposite kidney is normal.
- 3- Percutaneous nephrostomy
 - Indication: compromised cases with bad other kidney
 - Duration: till obstruction is treated
- 4- Pyeloplasty → a plastic operation to reduce the pelvis size
 - Indication: advanced cases of pelvic hydronephrosis
 - Advantage: provide adequate drainage of lowest part of the pelvis

Stricture of the ureter

Etiology

1- Congenital.

- At PUJO or at uretero-vesical junction (**pin-hole meatus**)

2- Traumatic.

- Follow open or closed injuries particularly operative or instrumental

3- Inflammatory

- a) Bilharzial → affects intramural and lower part of the ureter and less commonly opposite 3rd lumbar vertebra (may be bilateral)

- b) Tuberculous → affects whole ureter (usually multiple & bilateral)

4- Long-standing stone

5- Neoplastic.

- a) Carcinoma of the ureter

- b) Invasion of ureter by carcinoma of uterus, colon or rectum.

• In Egypt, bilateral hydronephrosis is commonly due to bilateral Bilharzial ureteric stricture → chronic renal failure

Clinical picture

- 1- Renal aching pain (due to renal pelvis distension)
- 2- Progressive hydronephrosis.
- 3- Recurrent attacks of pyelonephritis.
- 4- Chronic renal failure (in bilateral Bilharzial stricture)

Investigations

Laboratory

- Blood urea, serum creatinine and serum electrolytes.

Radiological

- 1- Ultrasound → hydronephrosis

- 2- IVU

Determine stricture site & degree of functional capacity of kidneys

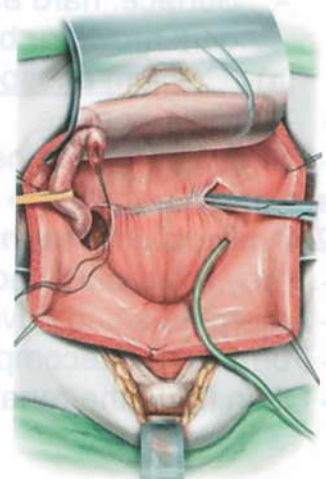
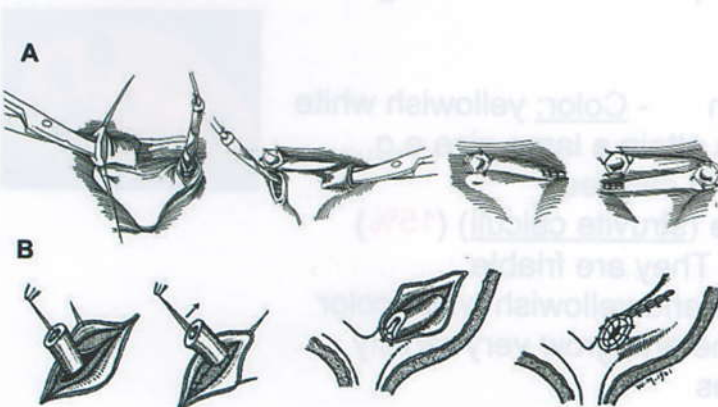
- 3- Retrograde and antegrade pyelography



Treatment

Mainly surgical

- 1- Stricture of the upper 2/3 → excision and end-to-end anastomosis
- 2- Stricture of the lower 1/3 → uretero-vesical implantation



Urinary stones

Etiology & Predisposing factors

A- Urine stasis

- As in BPH and strictures → allow stones to form.
- Stasis also predisposes to infection.

B-Excess normal constituents in urine

i) Low urine volume → raises its concentration.

ii) Excess urinary excretion of calcium:

- 1- Idiopathic
- 2- Hyperparathyroidism
- 3- Prolonged immobilization

iii) Excess urinary excretion of uric acid

- 1- Gout
- 2- Chemotherapy
- 3- Purine-rich food as meat, liver & kidney

iv) Excess urinary excretion of oxalates

- 1- Idiopathic hyperoxaluria
- 2- Excess dietary intake of strawberries, green leafy vegetable & tea
- 3- Loss of terminal ileum, e.g., by resection or Crohn's disease

C- Presence of abnormal constituents in urine

1- Infection

- Produces epithelial desquamation upon which calculi deposit.
- Infection with urea-splitting organisms → urine alkalization → favours formation of phosphate stones.

2- Foreign bodies → act as a nidus for stone formation

- Such as non-absorbable sutures, ureteric stents or catheters

3- Vitamin A deficiency (rare)

- Cause hyperkeratosis of urothelium → epithelial debris acts as nidus for stone formation.

4- Cystinuria → cystine stone formation

Composition of urinary calculi

1- Calcium calculi (75%) → majority of urinary stones

(Either calcium oxalate, calcium phosphate or a mixture of the two)

a) Calcium oxalate calculi (the most common)

- Are radio-opaque.
- Surface: hard and spiky → injure epithelium → bleeding → dark brown or black color

b) Calcium phosphate stones

- Are radio-opaque
- Surface: smooth
- Color: yellowish white
- They grow in alkaline medium and can attain a large size e.g., **staghorn** calculus → fills renal pelvis and calyces

2- Ammonium magnesium phosphate (struvite calculi) (15%)

- They are radio-opaque
- They are friable
- Amorphous and have smooth surfaces and yellowish white color
- Form in foul decomposing alkaline urine and grow very rapidly
- Often filling the renal pelvis and calyces

Incidence

- Common → affects **10-20%** of population at some stage of life
- Males are affected more than females.
- Occur mainly in middle aged persons but no age is immune
- 2/3 of patients have 1 or more recurrences within following 8 years
- Normal urine flow → expels crystals before they coalesce and grow to form stones



3- Uric acid calculi (7-9%)

- Are radiolucent → not show on plain films
- Hard, smooth and golden yellow.
- Diagnosis: a) Ultrasound and CT
b) Excretory urograms → a filling defect

4- Cystine stones (1-3%).

5- Xanthine stones (very rare)



Clinical picture

A) Renal and ureteric calculi

- Asymptomatic in staghorn stone or a tiny stone in a calyx.
- Symptoms when a stone gets impacted → obstruction.
- Common sites for stone impaction are: i) pelvi-ureteric junction
ii) Pelvic brim as ureter crosses iliac artery
iii) Uretero-vesical junction

1- Pain (the main symptom)

Renal pelvic stone

- Character: Fixed dull aching pain
- Site: in renal angle
- Radiation: to the back or to the hypochondrium anteriorly
- Increases by: movement

Ureteric stone

- As the stone moves along the ureter → ureteric colic
- In upper 1/3 → colicky pain in the loin
- Radiates to: groin or testicles (males) or labia majora (females)
- Restlessness due to inability to obtain relief irrespective of position.
- In middle 1/3 → iliac pain (may simulate appendicitis)
- In lower end → Pain at end of micturition

- Referred to: the tip of penis
- Associated symptoms: frequency and strangury

2- Nausea and/or vomiting

- During an attack of pain due to irritation of coeliac ganglion.

3- Abdominal distension

4- Haematuria

- Gross or microscopic due to mucosal injury during stone migration.

5- Fever → rare unless there is UTI

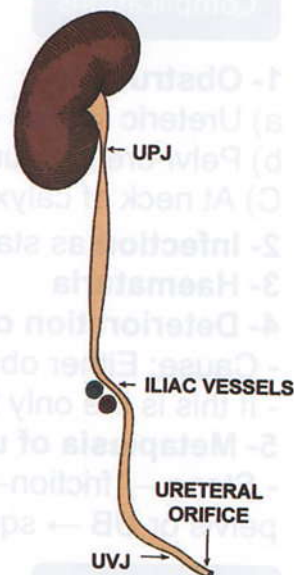
B) Urinary bladder calculi

1- Suprapubic bladder pain

- Referred to: tip of penis (or labia majora) or to the perineum.
- More prominent at day time (during micturition).
- Pain lessens during sleep or in recumbent position as stone moves away from the trigone

2- Frequency of micturition

- Cause: irritation of bladder mucosa and trigone.
- Timing: at first → only during day time (diurnal) but if cystitis develops → becomes diurnal and nocturnal.



3- Difficult micturition and interrupted urine stream during micturition

4- Acute urine retention

- Cause: obstruction of bladder neck by the stone.

5- Terminal hematuria

Few drops by end of micturition from abrasions of the trigone

C) Urethral calculi

1- Severe urethral pain, difficulty & interruption of micturition stream.

2- Retention of urine.

3- If a stone is in the anterior urethra it can be palpated

Complications

1- Obstruction

a) Ureteric stones → hydroureter & hydronephrosis

b) Pelvi-ureteric junction stone → hydronephrosis.

C) At neck of calyx → calycial dilatation (hydrocalyx)

2- **Infection** as stasis invites infection.

3- **Haematuria**

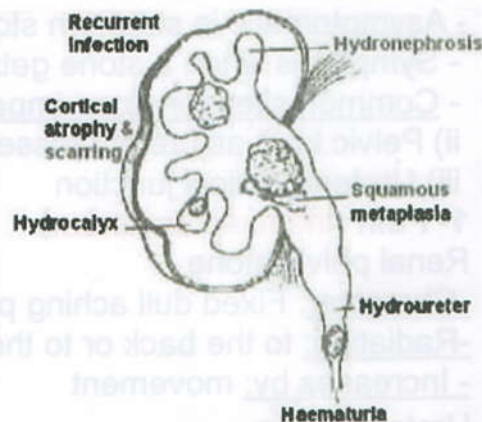
4- **Deterioration of renal function.**

- Cause: Either obstruction or infection

- If this is the only functioning kidney → renal failure

5- **Metaplasia of urinary epithelium**

- Stone → friction → chronic irritation of transitional epithelium in pelvis or UB → squamous metaplasia → squamous cell carcinoma



Investigations

Laboratory

Urine analysis

1- Haematuria (in at least 90%) → microscopic or

2- Crystals of same type that are forming the stone

Radiological

1- **Computed tomography (CT) (the first-choice)**

- CT UT without contrast → visualizes the smallest stones.

- Images can be reconstructed with computer software to produce high quality images of the whole urinary tract.

- Visualizes both radio-opaque and radiolucent stones

2- **Plain UT (KUB) (calculi show in 90%)**

- In descending order of density calcium phosphate is the densest, followed by calcium oxalate, struvite and cystine.

- Uric acid stones are radiolucent

3- **Ultrasonography**

- Advantages: a) simple

b) Visualize the kidney and the degree of back pressure changes.

c) Diagnose bladder stones and radiolucent calculi.

- Disadvantage: not reliable to visualize ureteric calculi



Intravenous urography (IVU) (mandatory in all cases)

Advantages:

- Confirms presence of stone and determines its position.
- Detects radiolucent calculi → appear as filling defects.
- Shows the cause of stone, e.g., stricture or BPH
- Gives idea about kidneys function & back-pressure effects
- Helps in choosing proper line of treatment for a particular stone



Differential diagnosis of radio opaque shadow on plain UT

- Urinary stones.
- GB stones (in **only 15%**)
 - Usually multiple, faceted, with signet ring appearance on RT side.
 - Lateral view → anterior to vertebral bodies near anterior abdominal wall (If renal stone → lie overlying the vertebral body, posteriorly)
- Pelvic phlebolith (a small spot of calcification in a pelvic vein)
 - Simulates ureteric stones, on IVU → lie outside the ureter.
- Calcified mesenteric LNs → multiple, mottled & take the line of root of mesentery (from RT iliac fossa to LT upper abdomen)
- Nephrocalcinosis
- Calcified renal tumours
- Fracture tip of transverse process of a lumbar vertebra.
- Calcified costal cartilage.
- Adrenal gland calcifications
- Appendicolith & FBs in intestine

Treatment

A) Treatment of renal colic

- Most cases are treated as outpatients.
- Hospitalization for patients with severe colic with persistent vomiting
- 1- Parenteral analgesics
 - NSAID as indomethacin or diclofenac.
 - Narcotic analgesics as opiates (in resistant cases)
- 2- Anti-emetics, e.g., metochlopramide.
- 3- IV fluids are given (according to normal patient's requirements) if vomiting is persistent
 - Forcing excess fluids during attack → flush out the stone, but if not → increase pressure in an obstructed system → increase pain
- 4- Antibiotics (if UTI is suspected)

B) Treatment of renal stones

I- Conservative medical treatment

Prerequisites

- Small stone < 0.5 cm (**90%** can pass spontaneously)
- No evidence of back pressure effect (hydronephrosis).
- No distal obstruction
- No evidence of infection.

- Remember the course of ureter to be able to suspect or exclude an opaque shadow as a ureteric stone

- Starts: opposite the tip of transverse process of L 1 (or L2) vertebra and then passes down along the tips of transverse processes of the following lumbar vertebrae.

Then crosses the pelvic brim opposite the sacro-iliac joint to enter the true pelvis

- At level of ischial spine, it turns medially to enter bladder

Method

- 1- High fluid intake (with or without diuretics)
- 2- Analgesics and antispasmodics.
- 3- Urinary antiseptics.
- 4- Regular follow-up to monitor stone descent.
- 5- Recovering any passed stone or gravel (either by filtering urine or by voiding in a clean container) → Stone analysis (allows planning for future therapy and prophylaxis)

II- Extracorporeal shock wave lithotripsy (ESWL)

Principle (performed without anaesthesia, except in children)

- High-energy shock waves that are focused from outside the body on the stone (after its visualization using x-ray or ultrasonic imaging)
- The stone is fragmented → pass out through the normal pathway.
- High fluid intake is maintained to facilitate passage of gravels.

Indication

All radio-opaque renal stones < 2 cm

Advantages

- 1- Non-invasive technique done as an outpatient procedure
- 2- Suitable for risky patients.
- 3- Successful for most radio-opaque urinary calculi.
(Uric acid calculi are radiolucent & may cause some difficulty)

Contraindications

a) Urological contraindications

- 1- Presence of distal obstruction (fragments will not pass).
- 2- Large stone (>2cm) except after debulking of stone by PCNL.
- 3- Renal insufficiency (kidney has no power to push fragments).
- 4- Acute episode of renal infection.

b) Non-urological contraindications

- 1- Pregnancy (X-ray exposure)
- 2- Bleeding diathesis

Complications

- 1- Transient attacks of haematuria caused by the passage of gravel.
- 2- Colicky ureteric pain during passage of fragments.
- 3- Ureteric obstruction from arrest of fragments in the ureter
- 4- Failure to fragment hard stones

III- Percutaneous nephrolithotomy (PCNL)

An endoscopic technique to fragment and remove renal stones

Indications

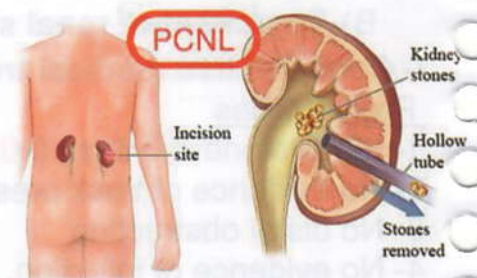
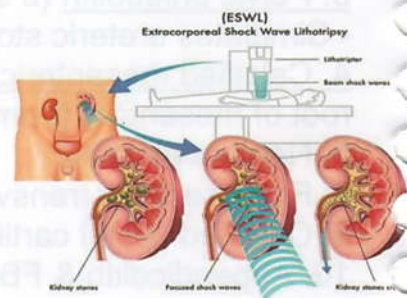
- 1- Stones >2cm
- 2- Urinary obstructive lesions
- 3- ESWL failure.

Contraindications

- 1- Bleeding diathesis
- 2- During pregnancy (X-ray)

Technique

1. Contrast material is injected up a ureteric catheter to visualize the pelvicalyceal system.
2. A needle is passed to the pelvis through which a guide-wire is inserted.



3. The tract is dilated over the guide wire to a size sufficient to accommodate the nephroscope
4. Stone manipulation.
 - Small stones < 1cm → directly extracted through the sheath.
 - Large stones are fragmented first through nephroscope by either ultrasonic, laser or electrohydraulic lithotripsy
5. After stone removal, a nephrostomy tube is left in situ for 48 hours to allow drainage of residual tiny fragments, urine and blood.

Advantages

- 1- Very small endoscopic incision (1cm).
- 2- Minimal operative and post-operative side effects.
- 3- Short hospital stays (about 3 days)

Complications

- 1- Kidney bleeding.
- 2- Extravasation of irrigating fluid into peritoneum or retroperitoneum
- 3- Residual stone fragments.
- 4- Injury to adjacent organs, e.g., the colon

IV- Open Surgery

5-10% of patients will be candidates for open surgery

Indications

- Contraindication or failed to ESWL or PCNL

Methods

1. Pyelolithotomy

- Definition: opening the renal pelvis and removal of the stone.

- Advantages: a) not cause any damage to renal parenchyma
b) Bleeding is minimal.

- The renal pelvis is reached by exposing the posterior surface of the kidney as it is the most posterior structure.

2. Nephrolithotomy

- Definition: incision through renal parenchyma to remove a stone.

- Indication: a) difficulty in exposing the renal pelvis
b) When stone is large and palpable through thin renal parenchyma.

3. Pyelonephrolithotomy (combines the above two procedures)

- Indication: branched calculi

4. Partial nephrectomy

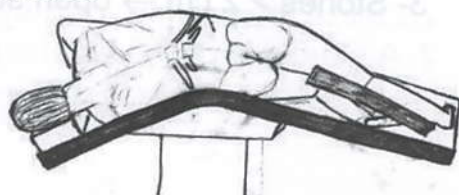
- Indication: a stone impacted in a hugely dilated lower calyx with narrowing of its neck.

5. Nephrectomy

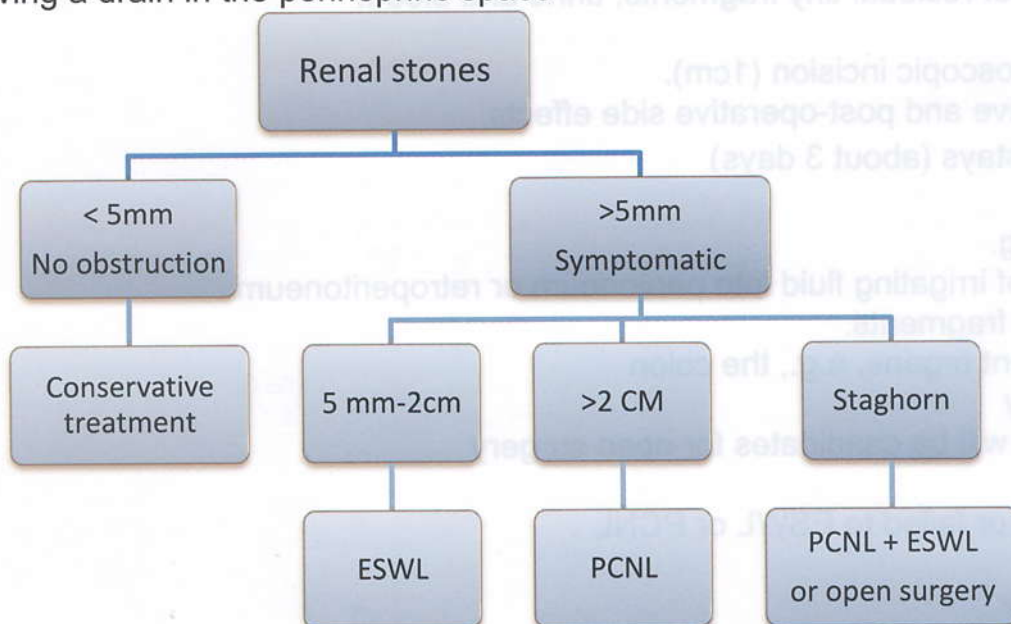
- Indication: the kidney is not functioning provided that the contralateral kidney possesses a good function.

Surgical access to the kidney

1. Do plain x-ray on the day of operation to confirm stone position
2. The patient is turned on his side (lateral position), with diseased side up and the bridge in the middle of the table is elevated to improve access.



3. The kidney is exposed by an oblique **lumbar incision of Morris**.
- This is a muscle cutting incision that starts at renal angle & carried downward & forwards parallel to and about 1 inch below last rib.
4. Muscles are divided then peritoneum is peeled forwards.
- Incision of the perinephric fascia exposes the kidney.
5. The stone is removed by incising renal pelvis, parenchyma or both
6. Incision in the pelvis is closed. The wound is closed in layers leaving a drain in the perinephric space



C) Treatment of ureteric stones

Conservative treatment follows same principles as for renal stones

Indications for intervention

- 1- Stones > 5 mm
- 2- Evidence of 2nd infection
- 3- Evidence of back pressure effect
- 4- Persistent pain.
- 5- Failure of conservative treatment

Types of intervention (according to stone site & size)

I) Upper third of ureter

- 1- Stone < 1 cm → ESWL with stone in place or stone is pushed up to kidney by a ureteric catheter then ESWL is used as in renal stone.
- 2- Stone > 1 cm → open surgery

II) Middle third of ureter

- Open surgery (the treatment of choice)
- Ureteroscopic extraction → dangerous and not advised.
- ESWL → very difficult and not efficient

III) Lower third of ureter

- 1- Stones < 1 cm → extracted by ureteroscopy (URS).
- 2- Stones < 2 cm → fragmented and extracted by ureteroscopy.
- 3- Stones > 2 cm → open surgery

Ureteroscopy (URS)

Technique

- 1- Cystoscopy to identify ureteric orifice.
- 2- Fine ureteroscope is passed up the ureter under vision.
 - a) Small stone → a wire basket passed through the ureteroscope and is removed under vision.
 - b) Large stone → fragmentation by ultrasound, laser or electrohydraulic lithotripsy under vision.

Complications of ureteroscopy

Perforation, avulsion or stricture of the ureter and infection

• Open Surgery (ureterolithotomy)

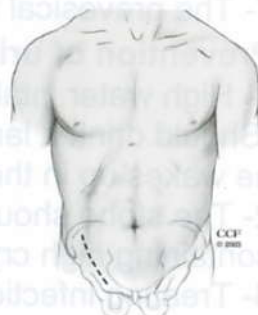
Indications

- 1- Stones >1 cm in upper ureter
- 2- Stones >2cm in lower ureter
- 3- Stones in middle ureter
- 4- Associated ureteric stricture
- 5- Failed URS or ESWL
- 6- Contraindication to URS or ESWL

Operation

1- Incision

- a) Stones in upper third → lumbar incision (as in kidney exposure)
 - b) Middle third & lower third above the level of the ischial spine → **Abernathy's** incision (a muscle cutting incision that starts above the anterior superior iliac spine and passes downwards and medially parallel to the lateral half of inguinal ligament)
 - c) Lower part (below the level of the ischial spine) → lower midline or Pfannenstiel's (transverse) incision
- 2- Plain x-ray just before the operation is important
 - 3- At any level, the ureter is exposed by an extraperitoneal route (peritoneum is not opened but is peeled medially)
 - 4- It is preferable to open ureter in a healthy area above the stone.
 - 5- The stone is removed by a ureterolithotomy forceps.
 - 6- A small ureteric catheter (6F) is passed downwards the ureter to check its patency and to exclude any stricture.
 - 7- The ureter incision is closed by fine 4-0 polygalactin (Vicryl) suture
 - 8- A drain is inserted and the wound is closed in layers.



D) Treatment of urinary bladder stones

I- Cystoscopic litholapaxy

- Indication: Stones < 2 cm.

- Method:

- 1- The stone is crushed cystoscopically either mechanically by **lithotrite** (technique is called litholapaxy) or by ultrasonic waves or electrohydraulic shock waves.
- 2- Fragments are then lavaged cystoscopically to outside the bladder by **Ellik evacuator**.

Due to high pressure in bladder during voiding, stones < 1 cm have no chance to stay in the bladder unless there is distal obstruction as in stricture or prostatic enlargement



II- Open surgery (cystolithotomy)

- Indications:

- 1- Stone > 2 cm
- 2- Multiple stones
- 3- Stone in a diverticulum (has thin wall, so easy to perforate).
- 4- Presence of another pathology needing surgery.
- 5- Failure of crushing the stone.

- Steps:

- 1- Low midline incision or a Pfannenstiel's transverse incision
- 2- The peritoneum is peeled upwards.
- 3- The bladder is identified by:
 - a) its trabeculated muscular wall
 - b) The presence of a plexus of veins over its surface.
- 4- The bladder wall is held between 2 bladder forceps & opened
- 5- The stone is extracted and the bladder is inspected for any associated pathology.
- 6- The bladder is closed in 2 layers with 2-0 polygalactin (Vicryl) after insertion of a suprapubic tube or a urethral catheter.
- 7- The prevesical space is drained and the wound is closed

Prevention of urinary stone recurrence

- 1- High water intake especially in hot weather
(Should drink a large glass of water before going to bed and again if he wakes up in the middle of the night)
- 2- The stone should be chemically analyzed → Restrict diet containing high crystals according to stone composition.
- 3- Treating infections and other causes of stone formation.
- 4- Follow up of stone formers to detect early recurrences.
- 5- Metabolic work-up to know etiology of stone.
- 6- Serum calcium and phosphorus to exclude hyperparathyroidism.
- 7- 24-hour urine collection for calcium, uric acid, oxalate and citrate.

Specific measures according to the type of the calculus

Calcium oxalate calculi

- 1- Reduce food that is rich in oxalates as chocolate, spinach, tomatoes and strawberry.
- 2- Alkalinize urine by citrates to inhibit crystallization of oxalates.

Uric acid calculi

- 1- Reduce food rich in purines as meat & organ meat (brain & liver)
- 2- Alkalinization of urine by citrates.
- 3- Allopurinol (Zyloric) 300 mg/day in patients with hyperuricemia → inhibits uric acid formation

Ammonium magnesium phosphate (struvite)

- 1- Aluminum hydroxide orally → restricts phosphate absorption.
- 2- Long term antibiotics → eradicate urinary tract infection.
- 3- Avoid indwelling catheters.
- 4- Increase urine acidity by oral administration of 1 g vitamin C daily

A person who formed a stone is liable to develop recurrent calculi.

Calcular anuria

Definition

A common serious and urgent problem in which there is arrest of urine flow (anuria) ^{2^{ry}} to stone obstruction of the ureter(s).

Etiology

- 1- Stone obstructing the ureter of an only functioning kidney (The other is nonfunctioning, surgically removed, or congenitally absent)
- 2- Bilateral renal or ureteric calculi

Clinical picture

Stage of onset

An attack of ureteric colic is followed by anuria (colic may be absent)

Stage of tolerance

- 1-Colic disappears and there are no major symptoms or signs.
- 2-Still there is no urine output
- 3- Urea and creatinine start to rise.

Stage of uraemia (renal failure)

Later, clinical picture of uraemia will follow within a few days.

Investigations

Laboratory

Blood urea, creatinine and potassium → elevated to varying degrees according to the time of presentation

Radiological

- 1- A plain x-ray → may show a radio-opaque stone
- 2- Abdominal ultrasound → reveals a distended pelvi-calyceal system on affected side

Treatment

It is urological emergency

- 1-Fixing a urethral catheter then a trial of forced diuresis using **40 g** furosemide in **100 ml** Saline over **2 hours**
- 2- Drainage of the obstructed kidney as early as possible.
 - a) Under anaesthesia a cystoscope is inserted and a ureteric catheter is passed up the affected ureter past the stone (If it succeeds a large amount of urine is drained)
 - b) If the side of obstruction is not exactly known, both ureters should be catheterized)
- 3- If ureteric catheterization fails → do percutaneous nephrostomy under ultrasound or fluoroscopy guidance → allows urine drainage
- 4- After renal function improvement the patient is investigated and diagnosed & the stone is treated as mentioned earlier.

- It should be remembered that a large hydronephrotic kidney is usually not the side of acute obstruction as it is often nonfunctioning.

- IVU is contraindicated in cases of renal failure

-Differential diagnosis

Acute retention of urine (In cases of anuria, the urinary bladder is empty and not palpable)

- To clinically determine the side of obstruction, it is usually the side of recent ureteric or renal pain and the side with renal tenderness or guarding

- Following relief of urinary obstruction, the patient may have severe diuresis (pass several liters of urine /day) → should properly replace the lost large amounts of fluid & electrolytes

Benign prostatic hyperplasia (BPH)

Incidence

- 1- Enlargement of the prostate starts at the age of **40 years**.
- 2- By the age of **50** years, about **50%** of males have BPH.

Etiology

- The exact cause is not known
- It is suggested that hormonal imbalance between androgens & Estrogens (due to aging) → stimulates transition-zone prostatic cells to undergo hyperplasia.
- Development of BPH is dependent on the presence of a functioning testis with adequate testosterone production.

Pathology

Microscopic picture

Hyperplasia of glandular, fibrous & muscular elements

Gross picture

I- Changes in the prostate

- 1- The prostate is not divided into lobes but is actually divided into morphologically & functionally different zones:

- a) Anterior fibromuscular stroma
- b) Peripheral zone → origin of prostate cancer
- c) Central zone
- d) Transition zone (periurethral area) → the origin of BPH

- BPH starts by development of nodules that enlarge → compress urethra & rest of prostate.

- These nodules, later, form the main bulk of BPH tissue and the remaining compressed prostatic tissue forms a sort of surgical capsule within which the adenoma (nodular tissue forming BPH) is enucleated during surgery

- If hyperplasia is more prominent on:

- a) Both sides of urethra → described as enlargement of lateral lobes
- b) Central & grows up elevating trigone → middle lobe enlargement
- c) In some cases enlargement maybe trilobar.

II- Urethral changes

The adenoma compresses the urethra → elongation; narrowing and angulation → makes catheterization difficult

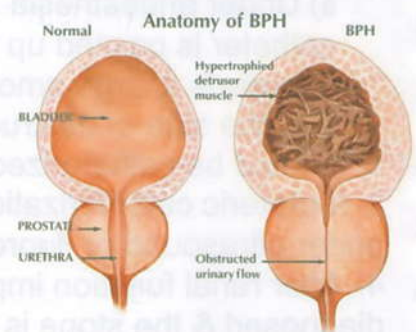
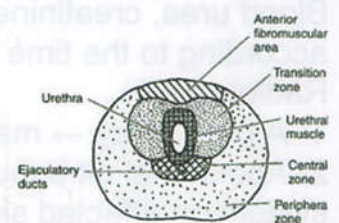
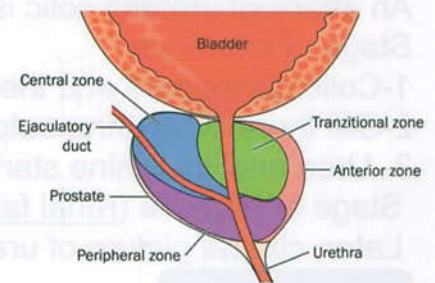
III- Urinary bladder changes

Suffers from chronic obstructive uropathy in the form of hypertrophy, trabeculation, sacculation, and diverticula formation → does not empty completely → post-voiding residual urine

IV- Ureters and kidneys

Later, unilateral or bilateral hydroureter & hydronephrosis

BPH is a normal aging process that affects most males → therefore, also called senile Prostatic enlargement



Clinical picture

Symptoms (include irritative & obstructive complaints)

1- May be asymptomatic

2- Frequency and urgency (the usual early complaints)

-At first, mainly nocturnal (wake up **2-3 times** by night to void)

Later → becomes progressive and occurs night and day.

3- Hesitancy (has to wait for a few seconds to start micturition), intermittent stream and terminal dribbling (difficulty to start, maintain & terminate micturition)

4- Weak and diminished caliber of urine stream.

5- **Acute retention** of urine (may be the 1st presentation)

- Precipitated by: excess fluid intake, diuresis, constipation, cold weather, alcoholic drinks, cystitis, prolonged confinement to bed, unrelieved sexual excitement, and drugs (e.g., diuretics, antispasmodics or anti-tussives)

-Very painful and needs urgent intervention

6- **Chronic retention with overflow incontinence**

-As obstruction progresses → more residual urine accumulates until bladder becomes over distended (may contain up to one liter urine)

→ The bladder turns atonic and patient involuntarily voids small amounts when intravesical pressure exceeds intraurethral pressure (The condition is painless & patient complains of incontinence)

7- **Haematuria**

An enlarged prostate → compress prostatic venous plexus at base of bladder → congestion → rupture of veins → gross haematuria

8- **Symptoms of complications** → bladder stone, cystitis, bladder diverticula or chronic renal failure.

Signs

1- Evidence of chronic renal failure may be present.

2- Neurological examination → to exclude a neurogenic bladder

3- Suprapubic mass (full bladder) may be felt.

4- **DRE** → prostate is felt enlarged, smooth and firm.

- Median sulcus is preserved & rectal mucosa can be moved over prostate (to DD from cancer prostate)

Investigations

Laboratory

1- Serum PSA (prostate specific antigen) → to exclude prostate cancer (Normal value is **0-4 ng/ml**)

2- Urine analysis, CBC, blood urea, creatinine and electrolytes

Radiological

1- Flowmetry

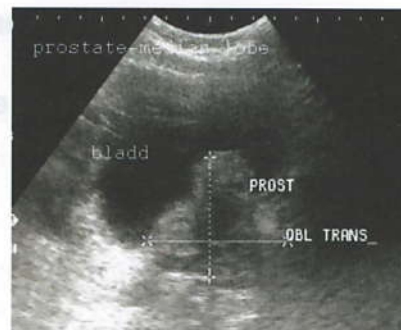
- Maximum flow-rate < 15 ml/s is indicative of obstruction.

2- Abdominopelvic ultrasonography

a) Visualize kidney changes

b) Measure post-voiding residual urine

- The severity of symptoms does not necessarily correlate with the size of the prostate.
- Any attempt to strain → prostate congestion → more obstruction (the patient has to relax in order to void)
- Commonest cause of neurogenic bladder is diabetic autonomic neuropathy



3- IVU shows the following changes:

- Smooth basal filling defect in the bladder
- Sacculation and diverticula.
- Post-voiding residual urine.
- Hydronephrotic changes of the kidneys.



4-Transrectal ultrasound (TRUS)

- Assess the size of the prostate.
- Guide prostatic biopsy if prostatic cancer is suspected



Treatment

A) Medical treatment and watchful waiting

- Indication: patients with minimal symptoms

Watchful waiting

- Avoid diuresis, constipation, wine, unrelieved sexual excitement and withholding micturition for long periods.
- Close follow-up with periodic Flowmetry and symptoms score.

Medical treatment

1) **Alpha-blockers**, e.g., tamsulosin

- Block alpha receptors in bladder neck & prostatic urethra → diminish outlet muscle tone (They improve particularly frequency & flow-rates)

2) **5-alpha-reductase inhibitors**, e.g., finasteride and dutasteride.

- Inhibit the enzyme 5-alpha-reductase that changes testosterone to active dihydrotestosterone in the prostate.

- Those who improve on this treatment, continued the drug for life

B) Minimally invasive procedures.

- Aim: relieve symptoms when drugs are not effective while definitive treatment is not suitable or contraindicated as in high risk patients.

- Include: a) Ablation with microwave and radiofrequency

b) Placement of a urethral stent.

C) Definitive surgical treatment

- The idea: remove the adenoma from inside the false capsule of compressed prostatic tissue.

- Indications: i) patients with marked symptoms

ii) Patients with complicated BPH as:

- 1-Recurrent acute retention episodes
- 2- Chronic retention with significant post-void residual urine
- 3- Gross haematuria
- 4- Bladder diverticula or stones
- 5- Recurrent UTI
- 6- Back-pressure effects on upper UT with renal impairment.

• BPH is a normal aging process that is not a disease by itself unless it causes obstruction

• BPH and prostate cancer can be present in the same gland

-Regular PSA evaluation for all men > 50 helps early detection of cancer prostate

• When complicated, the treatment of BPH is usually by TURP

- Techniques:

I- Endoscopic transurethral surgery

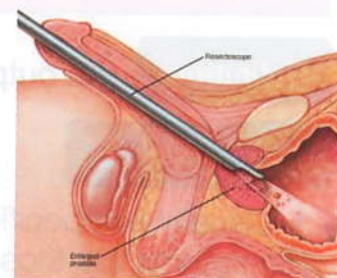
1- Trans-urethral resection of the prostate (**TURP**) (**the standard**)

Using a cysto-resectoscope the adenoma is removed trans-urethrally piece by piece, using an electrocautery

- Limitation: large adenoma → lengthy & hazardous procedure

2- Visual laser ablation of the prostate (VLAP).

3- Transurethral heat prostatic vaporization



II-Open surgical techniques

Indication: for exceptionally large adenomas.

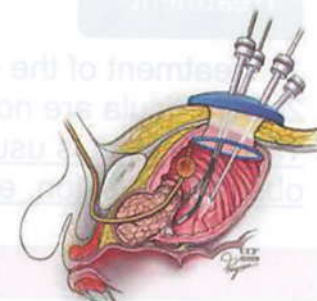
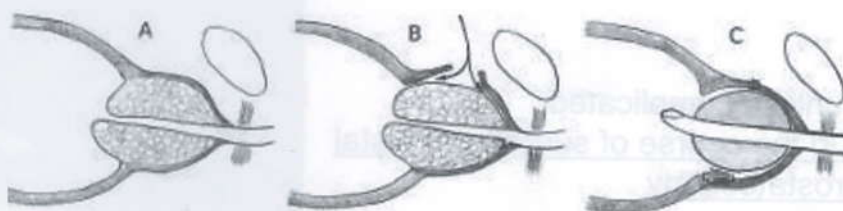
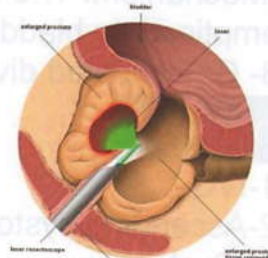
-Methods:

1- **Transvesical prostatectomy.**

- Indication: when there is an associated bladder pathology that requires surgery, e.g., diverticulum or big stone

2- **Retropubic (Millin's) prostatectomy.**

The retropubic space is exposed, but the bladder is not opened. A transverse incision is done directly through capsule of the prostate



-Complications of surgery

1- Bleeding (**the main problem**) → 1^{ry}, reactionary or 2^{ry}.

Clot retention may occur.

2- Retrograde ejaculation (occurs in **> 90%** of cases)

Cause: damage of the internal sphincter.

3-Infection → urethritis, cystitis or epididymo-orchitis

4- Bladder neck fibrosis and urethral stricture.

5- Urinary incontinence.

- True incontinence from sphincteric injury is rare (in 1/10,000)

-Temporary urge & stress incontinence for a few days are common.

6- **TURP syndrome**

-Incidence: a rare complication only with endoscopic resection

- Cause: absorption of irrigating hypotonic fluid during endoscopy

-Characterized by: hypervolaemia, dilutional hyponatraemia and intravascular haemolysis.

- Avoidance: The use of glycine (isotonic solution) for irrigation

Bladder diverticula

Definition An outpouching of the bladder wall

Clinical picture

- 1- Usually no specific symptoms indicative of diverticulum (Symptoms are those of the obstructive cause)
- 2- **Double micturition** (in large diverticula)
- Mechanism: The diverticulum fills with urine during micturition then empties into bladder → immediate desire to pass urine for a 2nd time.
- 3- Complicated diverticula → frequency, pain and haematuria.

Diagnosis

- 1- IVU
- 2- Ascending cystography
- 3- Cystoscopy

Confirm diagnosis & determine the cause

Treatment

- 1- Treatment of the cause.
- 2- Diverticula are not excised unless complicated.
The operation is usually done in the course of surgery for distal obstructive lesion, e.g., with prostatectomy

- It may be congenital, but most are acquired due to infravesical obstruction

Complications

- 2- Ureteric obstruction (rare)
- 2- Stones formed inside (due to stasis)



Urethral stricture

Etiology

- 1- Post-traumatic (**the most common cause**)
- 2- Post-gonococcal infection (affects bulbar urethra in **70%**)

Clinical picture

- 1- Difficult micturition.
- Patient has to strain to pass urine (in contrast to BPH)
- The stream is thin and weak.
- Terminal dribbling (as urine trickles from dilated proximal urethra)
- 2- Urethral discharge (because of infection)
- 3- Frequency due to incomplete bladder evacuation or cystitis
- 4- Acute retention
- Precipitated by excess fluid intake or by activation of local infection.
- 5- Chronic retention
In long-standing cases & causes bladder fullness and possibly dribbling due to retention with overflow

Incidence

Usually in young active males (rare in females)

Complications

- 1) Complications of obstructive uropathy as back pressure changes, infection, stone formation & renal failure.
- 2) 2^{ry} infection with periurethral abscess formation, spontaneous rupture or drainage → Urethral fistula

Investigations

1- Uroflowmetry

- Reveals obstructed flow characterized by straining

2- Ascending urethrography

Detects site, length, degree & shape of stricture

3- Urethroscopy

To see the stricture and assess its possible treatment

4- Sonography to assess the upper urinary tract

5- IVU to assess renal function and back pressure changes



Treatment

1- Endoscopic visual internal urothotomy

- Through a urethroscope & under direct vision, stricture is visualized and incised with a sharp knife blade → followed by regular dilatation.

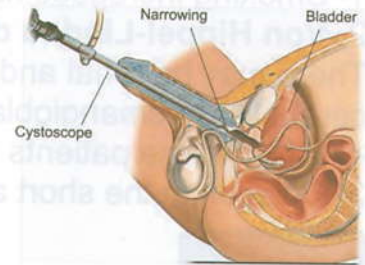
2- Surgical urethroplasty

- Indication:

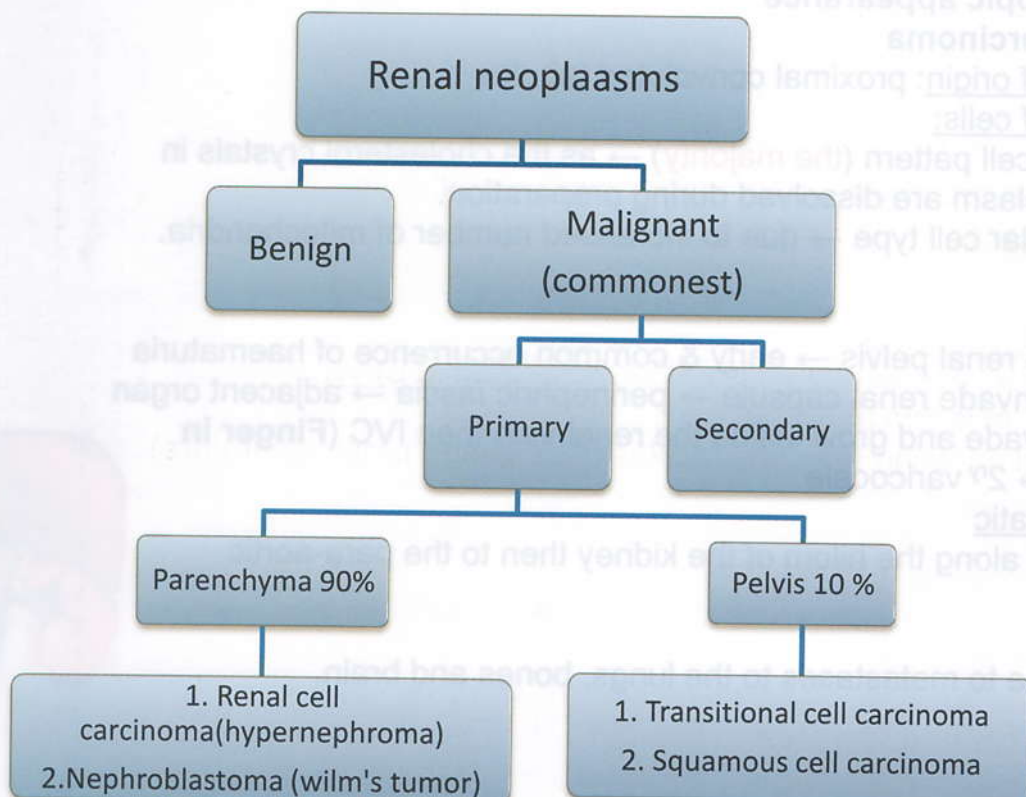
Bulbar & penile urethral strictures if previous measures fail

- Idea:

- Excise fibrous tissue of a stricture & do end to end anastomosis.
- If the stricture is long, a tube or a flap is constructed from penile skin, scrotum, or perineum & used to replace the narrow segment



TUMOURS OF THE URINARY TRACT



Renal cell carcinoma (hypernephroma)

Incidence

- Accounts for **75%** of all primary renal neoplasms.
- Male to female ratio is **2: 1**.
- The majority of patients are **40-60 years** of age.
- The tumour is usually unilateral (Bilateral in **2-3%** of cases)

Etiology

- 1- Smoking increases risk **two-folds**
- 2- **Von Hippel-Lindau disease** → higher incidence
There may be renal and pancreatic cysts, retinal angiomas, cerebellar haemangioblastomas and pheochromocytoma.
 - RCC in these patients may be multiple and bilateral.
- 3- Deletion in the short arm of chromosome 3

Pathology

Gross appearance

- 1- Commonly starts in one pole of the kidney.
- 2- The tumour color is **golden yellow** (due to its high lipid content)
- 3- Areas of haemorrhage and necrosis are common.
- 4- There is an apparent false capsule surrounding the lesion but the tumour actually invades the adjacent tissues beyond it.
- 5- Infiltrates the renal pelvis early in the course of the disease.

Microscopic appearance

Adenocarcinoma

- Cells of origin: proximal convoluted tubules

Types of cells:

- a) Clear cell pattern (**the majority**) → as the cholesterol crystals in the cytoplasm are dissolved during preparation.
- b) Granular cell type → due to increased number of mitochondria.

Spread

1- Direct

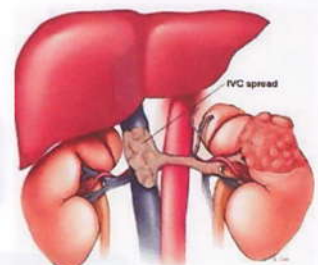
- Early to renal pelvis → early & common occurrence of haematuria
- Late to invade renal capsule → perinephric fascia → adjacent organ
- May invade and grow inside the renal vein then IVC (**Finger in glove**) → 2nd varicocele

2-Lymphatic

- To LNs along the hilum of the kidney then to the para-aortic nodes

3- Blood

Gives rise to metastases to the lungs, bones and brain.



Staging → TNM staging

T- primary tumor	
T1	Tumour < 7 cm, limited to the kidney
T2	Tumour > 7 cm, limited to the kidney
T3	Tumour extends into major veins or directly invades adrenal gland or perinephric tissues
T4	Tumour invades beyond Gerota's fascia
N - Regional lymph nodes	
N0	No regional lymph node metastasis
N1	Metastasis in a single regional lymph node
N2	Metastasis in > 1 regional lymph node
M - Distant Metastasis	
M0	No distant metastasis
M1	Distant metastasis

Clinical picture

- 1- Haematuria** (in **50%** of cases)
 - Characters: painless, recurrent, profuse and total haematuria.
 - Sometimes, blood passes as tubular clots taking shape of ureter
- 2- Pain** (in **40%** of cases), due to:
 - a) Stretch of the renal capsule by the neoplasm.
 - b) Passage of blood clots causing ureteric colic.
 - c) Infiltration of adjacent lumbar nerves causes referred pain.
- 3- Renal mass** → irregular hard renal swelling felt in **30%** of cases
- 4- Metastases** → may be 1st presentation (as to pulmonary or bone)
- 5- Non-specific symptoms**
 - As fever, night sweats, weight loss or anaemia.
- 6- Secondary varicocele**
 - Rapidly enlarging & does not empty by scrotum elevation
- 7- Systemic (para neoplastic) syndromes**
 - a) Hypercalcaemia (in **5%** of cases) due to:
 - i- Secretion of a parathormone-like substance by the tumour or
 - ii- Due to the presence of bone metastases.
 - b) Polycythemia due to excess erythropoietin secretion by tumour

Investigations

For diagnosis

- 1- CT scan (of choice)**, It is accurate in detecting:
 - a) The exact site, size and consistency of the lesion.
 - b) Invasion of the renal capsule or any surrounding tissues.
 - c) LN enlargement & invasion of the renal vein or INC
- 2- IVU** may reveal:
 - a) Enlargement of the kidney.
 - b) Elongation, displacement, compression or amputation of a calyx
 - c) Displacement of the whole renal pelvis.
- 3- Renal sonography** → shows the tumour as a solid mass.

For staging: Chest x-ray & isotope bone scan → detect metastases

•With the wide use of imaging modalities, many RCC are detected incidentally
 - The classical triad of haematuria, pain & renal mass is present only in **10%** of cases and indicates advanced disease



Treatment

A) Localized disease

- **Surgery** is the only treatment that may achieve cure.
- It consists of either: i) radical (open or laparoscopic) or ii) Partial (nephron-sparing) nephrectomy
- A prerequisite of removing a whole kidney is to ensure that the contralateral one is adequately functioning.
- Radical nephrectomy entails removal of the kidney within its sheath of Gerota's fascia.

B) Advanced disease

- Unfortunately, RCC is chemo-resistant & radioresistant.
- Patients who have metastatic disease may receive:
 - i) Immunotherapy (using alpha interferon or interleukin-2) or
 - ii) Targeted therapy (new drugs targeting angiogenesis).

- It is recommended to expose & ligate vascular pedicle as a 1st step of nephrectomy for:
 1. Prevention of dissemination of malignant cells during manipulation
 2. To have good control over a very vascular tumour
 3. To be able to remove a malignant thrombus from IVC, if present.

Wilms' tumour (nephroblastoma)

Origin

Arises from embryonic nephrogenic tissue

Incidence

- The most common renal tumour in childhood (**10%** of childhood malignant tumours & **80%** of all genitourinary cancers in children **< 15 years** of age)
- Peak incidence is **3 to 4 years** (90% occur before the age of seven)
- Male to female ratio is **1: 1**

Pathology

Gross appearance

- Appears as a large, grey, sharply demarcated, apparently encapsulated mass.
- Areas of haemorrhage and necrosis.
- Renal pelvis invasion is late.
- Bilateral tumours are present in **5-10%** of cases.

Microscopic picture

- Consists of mixed:
 - a) Epithelial (primitive glomeruli and tubules) and
 - b) Connective tissue (cartilage, fat, smooth or striated muscles).
- The degree of differentiation varies from favourable histology (FH) to unfavourable histology (UH) where there are marked variations in nuclear size with hyperchromatism and excessive mitoses

Staging

- Stage I → Tumour limited to the kidney and completely excised
- Stage II → Tumour beyond the kidney and completely excised.
- Stage III → Residual tumour.
- Stage IV → Metastases.
- Stage V → bilateral tumors



Clinical picture

- 1- An abdominal mass (main presentation 90%)
- Character: smooth, firm and is confined to one side of the abdomen
- 2- Vague abdominal pain (in 1/3 patients) often associated with minor trauma and haemorrhage within the tumour.
- 3- Microscopic haematuria (in **50%** of cases)
- 4- Hypertension (in up to **60%**)
- Cause: encroachment on the blood supply → renal ischemia → excess renin production.

Investigations

Laboratory

- 1- CBC, liver and renal function tests.
- 2- Urine catecholamines → normal (elevated in neuroblastoma)

Imaging

- 1- Ultrasonography (**the initial study of choice**).
 - Distinguish solid renal masses from hydronephrosis.
 - Can detect small tumours and liver metastases.
- 2- CT scanning
 - Differentiate cystic from solid masses.
 - Detect involvement of adjacent structures or the other kidney.
 - Follow the tumour response to chemo and radiotherapy.
- 3- Chest x-ray → detect metastases

Treatment

A) Surgery

- 1- For operable tumours → radical resection of the affected kidney
- 2- For large unresectable lesions → preoperative chemotherapy usually shrinks the tumour then can be removed.
- Remaining tumour in nodes or adjacent tissues should be marked with surgical clips to facilitate direction of radiotherapy.

B) Postoperative treatment

- 1- If there is no residual tumour → adjuvant chemotherapy using:
 - a) Actinomycin D
 - b) Vincristine
 - c) Adriamycin
- 2- If there is residual tumour, radiotherapy is added

Prognosis

- Multimodal therapy improved the overall 5-year survival to **80%**.
- Early cases are usually cured.
- The younger the child, the better is the prognosis

Differential diagnosis

- 1- Neuroblastoma
 - a) childhood tumour that simulates nephroblastoma
 - b) Forms an irregular tumour which may cross midline
2. Renal swellings → the commonest is hydronephrosis as a result of congenital PUJO



Surgical excision remains the cornerstone of therapy in all children with Wilms' tumour

Tumours of the renal pelvis

Transitional cell carcinoma (TCC)

Incidence

Accounts for **7%** of renal tumours

Predisposing factors

- 1- **Smoking** (the **main cause**) → increase in the risk 4-folds
- 2- Exposure to aniline dyes for workers in petroleum, rubber & textile industries.
- 3- Cyclophosphamide.

Pathology

- Origin: arise from the transitional epithelium that lines renal pelvis, ureter & urinary bladder → collectively called urothelial tumours
- Grade: vary from low-grade to highly anaplastic malignancies
- A patient with a TCC of renal pelvis is likely to develop same lesion in the ureter, bladder or contralateral renal pelvis because of either:
 - 1- Multicentricity of this neoplasm (the whole urothelium is predisposed to malignancy by a certain carcinogen)
 - 2- Transluminal spread of shed malignant cells

Clinical picture

- 1- Gross or microscopic haematuria (in **90%** of cases)
- 2- Obstruction causes renal pain and/or swelling (**hydronephrosis**)

Investigations

- 1- Intravenous urography (IVU)
 - Shows: a) fixed irregular radiolucent filling defect or b) Non-visualization of part or all of the collecting system.
- 2- CT scan
 - Differentiate renal parenchymal from renal pelvic tumors.
 - It can visualize LN deposits.
- 3- Cytology (microscopic examination of urine for malignant cells)
- 4- Flexible uretero-renaloscopy
 - A flexible endoscope is introduced from the bladder to inspect the whole upper tract (Any lesion visualized is biopsied)

Treatment

The classic therapy is nephroureterectomy with excision of an adjacent cuff from the bladder

Nephroureterectomy has two indications:

- 1- TCC of renal pelvis
- 2- Renal tuberculosis if the kidney is too damaged to respond to antituberculous therapy

Squamous cell carcinoma of renal pelvis

- Occurs on top of leukoplakia.
- Often associated with infection & calculus disease
- It forms a nodular ulcerated mass, which invades the parenchyma.
- Surface implantation does not occur.
- C/P:
 1. haematuria
 2. In late cases, severe pain (due to extrarenal spread)
- Has a poor prognosis



Urinary bladder cancer

Incidence

- The commonest urologic malignancy in Egypt
- Male to female ratio **4:1**
- Peak age incidence between 4th & 5th decades

Classification

- According to the histological type into:
 - 1- Squamous cell carcinoma (SCC)
 - 2- Transitional cell carcinoma (TCC)
 - 3- Adenocarcinoma.

Predisposing Factors

In the past, SCC accounted for the vast majority of bladder cancers, but currently, TCC is the commonest

Squamous cell carcinoma (SCC)

A) Bilharzial SCC

- Occurs on top of long-standing Bilharzial cystitis that is complicated by 2^{ry} bacterial infection (exact carcinogenesis mechanism is not known)

B) Non-Bilharzial SCC

- Occurs with longstanding bladder irritation that is complicated by 2^{ry} bacterial infection as in long-standing bladder stones

Transitional cell carcinoma (TCC)

Similar to TCC of the renal pelvis

1- Cigarette smoking (the most important)

Increases risk **four-folds**

2- Industrial carcinogens such as aniline dyes as petrol, leather, rubber & textile industries due to exposure to aromatic amines

3- Cyclophosphamide (chemotherapeutic drug) → **nine-fold** increase in risk.

4- Pelvic irradiation → **Four-fold** increase in risk noted in women treated for cervical malignancy.

Pathology

Alterations of the urothelium

- 1- Squamous metaplasia (the normal transitional epithelium is replaced by a mature, non-keratinizing squamous epithelium)
 - 2- Leukoplakia (mucosal well-defined, thick & raised white patches)
- Microscopically there is squamous metaplasia and marked keratinization with cellular atypia and dysplasia.

Both squamous metaplasia and leukoplakia are precancerous lesions for SCC

	Squamous cell carcinoma	Transitional cell carcinoma.
Gross appearance	- Nodular solid mass or a malignant ulcer on top of a large mass (fungating mass). - May arise from any part of UB	The common sites are: the base, trigone & around ureteric orifices. - May be single or multiple. - Form papillary villous growths
Microscopic appearance	- Composed of keratinized cells showing features of malignancy (as hyperchromia & multiple mitoses) and usually contains concentric aggregates of cells that are called squamous pearls (cell nests)	- Composed of multiple layers of transitional cells showing features of malignancy. - Stage of carcinoma in situ → malignant TCC cells without BM invasion.

Spread

1- Direct → to the wall of bladder and then may infiltrate adjacent organs as seminal vesicles & prostate in males or vagina in females

2- Lymphatic → to the iliac lymph groups.

3- Blood borne (common with TCC but is delayed with SCC)

TNM staging

T- primary tumor	N- regional LN	M- distant metastasis
- Tis: carcinoma in situ - T1: invades lamina propria - T2: invades muscle layer - T3: invades perivesical layer - T4: invades surrounding structures	- N0: No lymph node metastasis. - N1: LN metastasis.	- M0: no distant metastasis. - M1: distant metastasis

According to degree of cellular differentiation, TCC-is classified into:

- Grade I → well differentiated.
- Grade II → moderately differentiated.
- Grade III → poorly differentiated

Diagnosis

1-Haematuria (the earliest symptom in **TCC**)

- Character: painless intermittent haematuria.

2- Dysuria with recent exaggeration of cystitis (with SCC)

- There is burning micturition, frequency and pyuria.

3- Necroturia (the passage of pieces of whitish tissue)

- Occur in large necrotic tumour.

4- Signs of renal failure

-If tumor invades both ureters → back pressure on both kidneys

5- Manifestations of distant metastases (in advanced stages)

- Such as bony aches 2^{ry} to bone metastases.

6- DRE & bimanual examination → may detect presence of bladder mass & can assess size, site & tumour mobility

Investigations

1-IVU

-The tumour appears as an irregular filling defect

2- Abdominal ultrasonography → assess bladder mass

3- CT scan

(the best diagnostic tool for preoperative tumor staging)

4- Urine cytology

-Very useful in patients presenting with CIS or high-grade TCC

5- Cystoscopy and biopsy (the definitive diagnostic tool)



A) Squamous cell carcinoma

- By radical cystectomy (SCC is resistant to chemo & radiotherapy)
- There is no role for partial cystectomy.
- In males → resect the whole bladder with its perivesical fat, prostate, seminal vesicles & lower ends of both ureters, in one mass.
- In females → resect UB, uterus, tubes & anterior vaginal wall
- In both sexes → Block dissection of internal & external iliac and obturator LNs
- Urinary diversion is then performed.

B) Transitional cell carcinoma

I- Superficial TCC (Tis and T1) → depth of infiltration does not reach the muscle layer

1- Transurethral resection of the tumor (TURBT) → excise the tumour with underlying muscle plus multiple random biopsies to detect carcinoma in situ

- Regular check-up cystoscopy for 5 years to detect recurrence.

2- Postoperative intravesical chemotherapy and immunotherapy (by BCG vaccine) → used to prevent recurrence.

3- Radical cystectomy in cases with multiple recurrences

II- Muscle-invasive TCC (T2, T3 and T4)

1- Radical cystectomy (similar to that for SCC)

2- Radiotherapy

Reserved to those unfit for or refusing surgery as results are inferior

3- Chemotherapy

For metastatic disease or those with positive surgical margins

- Combination chemotherapy formed of methotrexate, vinblastine, Adriamycin and cis-platin "**MVAC**" is the **standard** protocol for TCC

Urinary diversion following cystectomy

Choice of method depends upon condition of patient & surgical experience

1- Ureterocolic anastomosis

- Operation: The two ureters are anastomosed to the sigmoid colon.

- Advantage: simple, rapid & the patient is continent.

- Disadvantages:

a) Recurrent upper UTI and deterioration of renal function

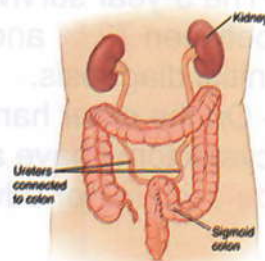
b) Absorption of chlorides in urine by colonic mucosa →

Hyperchloraemic acidosis

c) Hypokalaemia.

d) Formation of ammonia from the urea in urine → Encephalopathy if the liver function is compromised

e) Development of cancer colon in **30%** of patients after **10 years**

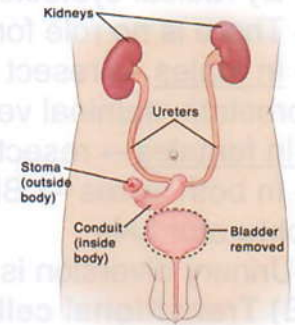


2- Ileal conduit

-Operation:

1. A segment of terminal ileum is isolated with intact blood supply.
2. The two ureters are implanted in the ileal segment.
3. At one end the ileal segment is closed & fixed at the other end to the skin as an ileostomy.
4. A special appliance is applied to the ileostomy to collect urine as this type of diversion is noncontinent

- Advantages: Ascending infection & renal function deterioration are less common than with ureterocolic anastomosis.



3- Rectal bladder

- Operation:

1. The sigmoid colon is divided above the recto-sigmoid junction.
2. The proximal end is brought out as a colostomy.
3. The distal end is closed & two ureters are implanted in rectum, which will now work as a urinary bladder.

- Advantages: 1. The patient will usually be continent for urine there
2. No hyperchloraemic acidosis.

- The main disadvantage: the presence of permanent colostomy.

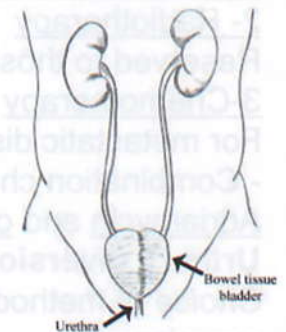


4- Continent orthotopic urinary diversion

- The standard urinary diversion nowadays whenever feasible, as it preserves the normal pattern of urination

-Operation:

1. Separate a segment of bowel with intact blood supply
2. Change its configuration from a tube to a sac so that it can accommodate and store urine.
3. The ureters are implanted in the new bladder & the base of this new bladder is anastomosed to the urethra..



Prognosis

-The 5-year survival for superficial TCC treated with TURBT ranges between **72 %** and **90%** depending on the tumor grade at the time of initial diagnosis.

- On the other hand, muscle invasive tumors managed with radical cystectomy have a **40-50%** survival rate for stage T2 which drops **below 30%** for advanced stages.

Prostate cancer

Incidence

- The most common solid organ cancer in men worldwide
- The prevalence shows a constant rise over successive decades starting at the age of **50 years**

Predisposing factors

1- Family history

- The relative risk is greatly increased if one or more first-degree relatives have the disease (father or brothers).

2- Race

- African Americans have the highest incidence while native Asian populations have the lowest incidence.

3-Rising age

Pathology

Gross picture

- Origin: Arise from the peripheral zone beneath the capsule (in the majority of neoplasms **75%**)
- The tumour appears as hard infiltrating pale nodule.

Microscopic picture → **95%** are adenocarcinomas.

Spread

1- Direct invasion

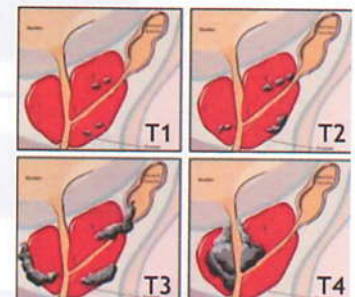
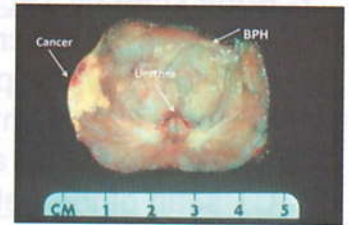
- Involves the seminal vesicles and surrounding tissues.
- Rectum is rarely invaded as fascia of Denonvillier acts as a barrier
- Ureteric obstruction may also occur.

2- Lymphatic spread

- To internal, external & common iliac LNs then to para-aortic LNs
- The obturator lymph nodes may also be involved.

3- Blood spread

- To bones in **90%** especially lower vertebrae, neck of femur, pelvic bones & ribs.
- The preference of pelvic & lumbar vertebrae is attributed to possible reversal of blood flow from the vesico-prostatic plexus to emissary veins of the pelvic bones.
- Metastases are usually osteoblastic.
- Rarely in the lungs or the liver.



Staging system TNM

T-primary tumor	N-regional lymph node	M-distant metastasis
T1: Clinically impalpable tumor.	No: no regional lymph node metastasis.	Mo: No evidence of distant metastasis.
T2: Tumor confined within the prostate.	N1: Regional lymph node metastasis	M1: Distant metastasis
T3: Extension through the prostatic capsule.		
T4:Tumor invades adjacent structures		

Investigations

Laboratory

1- Prostate specific antigen (PSA)

- The most useful marker for diagnosis and for follow-up.
- The normal value is **0-4 ng/ml**.
- An elevated level suggests prostate cancer but may be caused by other prostatic conditions such as prostatitis and BPH
- A markedly elevated **>20 ng/ml** suggests disseminated disease.

2- Acid phosphatase

- Raised in **70%** of patients with extracapsular or metastatic disease.
- Raised in many other conditions as Gaucher's disease, Paget's disease of bones & with breast and gastric cancer

3- Alkaline phosphatase → raised with bony or hepatic metastases.

4- Blood picture, blood urea and creatinine.

Imaging

1- Trans-rectal ultrasonography (TRUS)

- Give an idea about the extent of lesion & used to guide biopsy.

2- Bone scan by Technetium 99m

- Detect areas of increased bone activity
- Metastases will appear as hot spots.
- Not specific and any abnormality should be compared with the clinical examination and plain radiography.

3- CT scan of the pelvis → detect pelvic LN deposits.

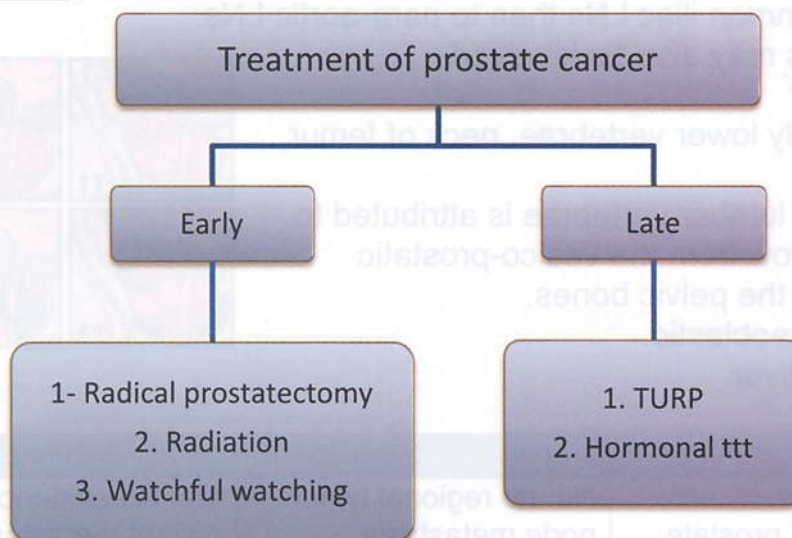
4- Plain X-ray of the chest, pelvis and spine

- May detect metastases which are usually osteoblastic

Biopsy (guided by trans-rectal ultrasound) to establish diagnosis

Indication: patients with suspected prostate cancer (due to elevated PSA or abnormal DRE findings)

Treatment



Treatment of prostate cancer depends on:

1. Local extent of the disease
2. The presence of metastases
3. General condition of the patient

1- Radical prostatectomy

- Indication: early localized disease in a fit patient.

- Operation: a) removal of prostate & seminal vesicles together with block dissection of obturator, internal and external iliac LNs

b) The bladder is then anastomosed to the urethra.

- Approach: open surgery (through retropubic) or laparoscopic

- Complications: a) urinary incontinence (in 5%)

b) Erectile dysfunction (in 30%)

2-Radiation therapy

- Indication: a) for localized disease and for locally advanced cancer

b) Palliative for symptomatic bone metastases → relieves pain.

- Method & Dose: External beam radiation therapy is applied giving high doses of around 70,000cGy over 5 weeks.

- Side effects:

a) Bladder irritation → frequency of micturition & urgency

b) Rectal irritation → diarrhoea or bleeding per rectum

c) Erectile dysfunction.

3-Hormonal therapy

- Indication: a) advanced/metastatic cases (mainstay of treatment)

b) For patients unwilling or are unfit for surgery or radiotherapy

- The rationale is that prostate cancer is androgen-sensitive tumour.

- Methods for androgen ablation:

a) Bilateral orchiectomy

- Acts by removing the main source of androgens.

- Disadvantages: loss of libido and erectile dysfunction.

b) Luteinizing hormone-releasing hormone (LH-RH) agonists

(e.g. leuprolide acetate)

- Action: initial rise of the level of testosterone for 4-6 weeks then a drop to castrate levels through their effect on the pituitary gland.

- They give equal results to castration & have same side effects.

c) Antiandrogens (e.g. flutamide).

- Action: block testosterone receptors by competitive inhibition.

- They are used as adjuvants to castration or LH-RH agonists.

d) Estrogens

- Act as anti-androgens.

- Major disadvantage → predispose to DVT

4- Transurethral resection of the prostate (TURP)

- Additional for advanced cases with bladder neck obstruction

5- Expectant management

Watchful waiting for small well-differentiated tumours in elderly men particularly if their anticipated life expectancy is < 10 years

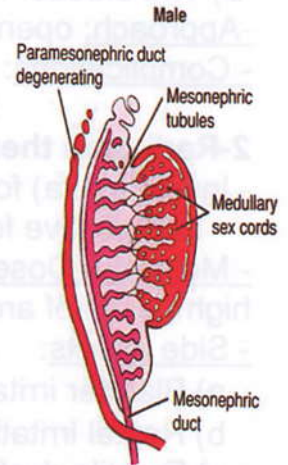
Prognosis

- Many small localized well-differentiated tumors will not progress and will not affect life expectancy
- With tumours localized to the prostate, the 10-year prostate cancer-specific survival is around 75%.
- If metastases are present, the 10-year survival rate drops to 10%.

The testes and scrotum

Development of the testes

- Develops from mesodermal genital fold behind peritoneum & below the developing kidney → receives its blood vessels and nerves high in the abdomen.
- The testis descends downwards pulling with it its blood & nerve supply and fold of peritoneum → **the processus vaginalis** → its distal portion persists as the **tunica vaginalis**
- As testis reaches the lower part of the abdomen, it unites with some of the mesonephric tubules → give the vasa efferentia.
- The Wolffian duct → develops the epididymis & vas deferens
- Normally, the testis reaches the scrotum just before birth.
- Descent of the testis depends upon:
 - a) Differential growth of the body (cranial segment grows faster than caudal segment)
 - b) Maternal chorionic gonadotrophins and pituitary gonadotrophic
 - c) Contraction of the gubernaculum (a fibromuscular tissue extending from bottom of the scrotum to lower pole of the testis)



Incompletely descended testis (undescended testis)

Incidence

- 1- Affects **1 %** of all males
- 2- Commoner on right side
- 3- Bilateral in **20%**
- 4- Higher incidence in premature

Predisposing factors

- 1- Mechanical factors:
Shortness of spermatic vessels or vas deferens
- 2- Deficiency of gonadotrophic hormones

Site

The testis may arrest anywhere along the normal line of descent:

- 1- In the abdomen (intra-abdominal).
- 2- In the inguinal canal
- 3- At the external ring
- 4- At the neck of the scrotum (high scrotal).

Clinical picture

- 1- Absence of one or both testes is usually discovered by the mother or the attending doctor (present since birth)
- 2- The ipsilateral side of the scrotum is empty & underdeveloped.
- 3- The testis is palpable if it lies high in the scrotum.
- 4- A testis that lies in the inguinal canal may be felt with difficulty. (Testis identified by giving characteristic sickening sensation when compressed)
- 5- An associated hernia may be present.

- Cryptorchidism means bilateral undescended impalpable testes

Congenital anomalies of the testes

1. Malescent of the testis
 - a) Incompletely descended testis (undescended testis).
 - b) Ectopic testis.
2. Testicular agenesis (rare).
3. Supernumerary testis (rare).

Complications

1-Depressed spermatogenesis

- Effective spermatogenesis requires a temperature less than that of body core → testes are normally placed outside abdomen in scrotum
- An undescended testis is exposed to body temperature → affects spermatogenic cells
- Up to **2 years**, the testis remains almost normal, after that, the higher temperature leads to development retardation
- At puberty, irreversible destructive changes in the germinal epithelium occur (sterility occur in bilateral cases)
- On the other hand, hormonal function of the testis remains normal, → 2^{ry} sexual characters develop normally (even in bilateral cases)

2- Increased liability to testicular malignancy

- Especially in intra-abdominal type.
- This is genetically determined & may affect the patient even after a successful orchidopexy.

3- Associated oblique inguinal hernia (in > 90% of cases)

4- Increased incidence of trauma (especially inguinal canal type)

5- Torsion of the spermatic cord 6- Psychiatric disturbances

Differential diagnosis

1. Ectopic testis
2. Retractable testis
3. Testicular agenesis.
4. Atrophy of the testis, e.g., after mumps orchitis.
5. Hermaproditism (should be excluded in bilateral cases)

Investigations

Laboratory

Hormonal assay (LH & testosterone hormone)

- To exclude anorchia in bilateral cases.

Radiology → Ultrasonography and CT scan

- Localize the site of the testis when it is not felt.

Laparoscopy → detects an intra-abdominal testis

Treatment

Surgery → **Orchidopexy** (the standard treatment)

- **Timing**: before the age of 2 years.

- **Operation**: 1. An inguinal incision is made.

2- An associated hernia sac is dissected & transfixed at internal ring

3- Mobilize vas deferens & testicular vessels from surroundings

4- The empty half of the scrotum is stretched to prepare a scrotal bed for the testis.

5- The mobilized testis is fixed in the scrotum to avoid retraction, commonly by placing it in a dartos pouch created between the skin of the scrotum and the dartos muscle.

Management of difficult cases

- **Cause of difficulty**: short testicular artery.

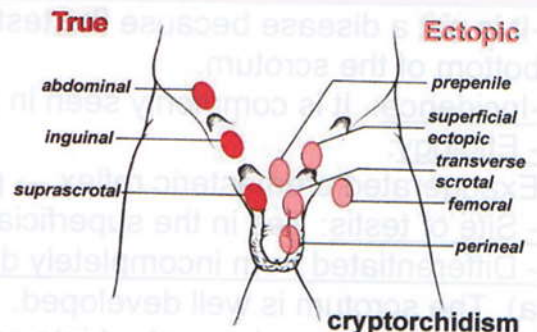
- **Management by either**:

1- **Staged orchidopexy** → testis is brought down in multiple stages

2- **Microvascular technique** → Division and re-anastomosis of the testicular vessels at a lower level to the inferior epigastric vessels.

3- **Orchidectomy**

Only if other testis is normal & undescended testis is atrophic.



Ectopic testis

-**Pathogenesis:** testis passed out of external inguinal ring then deviated outside normal route to a subcutaneous ectopic position.

-**Etiology:** the pull of an accessory gubernaculum.

- The possible sites: of

- (a) Inguinal region (**most common**)
- (b) Perineum
- (c) Root of the penis
- (d) femoral triangle

-**C/P:** 1- Empty scrotal compartment since birth.

2- The testis is felt in one of the above-mentioned ectopic sites.

The most frequent position is the superficial inguinal pouch.

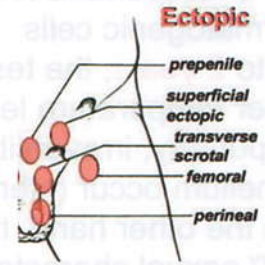
3- The hormonal and the spermatogenic functions are normal

DD: Undescended testes

(Being subcutaneous, the testis is easily felt and bulges more if the muscles contract while an undescended testis disappears because it is usually deep in the inguinal canal)

-**Treatment:**

Surgical replacement of testis in corresponding scrotal compartment



Retractile testis

-It is not a disease because the testis has descended normally to the bottom of the scrotum.

-**Incidence:** It is commonly seen in young children

- **Etiology:**

Exaggerated cremasteric reflex → pull testis up on contraction

- **Site of testis:** lies in the superficial inguinal pouch.

- **Differentiated from incompletely descended testis by:**

- a) The scrotum is well developed.
- b) The testis can be pushed into scrotum by sliding fingers down the inguinal canal, especially if child squats & draws the thigh to the abdomen, or if the child is examined during sleep in a warm room.

- **Diagnosis:**

If testis brought down to touch bottom of scrotum at any time

- **Treatment:** not required (reassure parents that the condition will be cured spontaneously)

Torsion of the spermatic cord (torsion of the testis)

Definition

Torsion of testis & epididymis around the axis of spermatic cord

Incidence

- Uncommon surgical emergency
- Most prevalent in adolescents

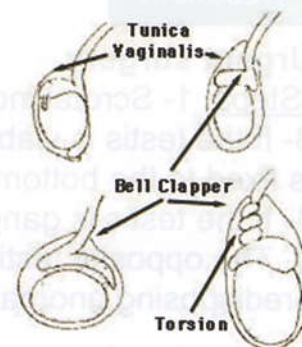
Etiology

Predisposing factors

- 1- Imperfect descent of the testis (incomplete or ectopic).
- 2- Wide tunica vaginalis that surrounds the whole testis → testis is suspended loosely inside its cavity (**bell-clapper deformity**).

Precipitating factors

A minor trauma or straining at defecation (spontaneous torsion sometimes occurs)



Pathology

- The testis rotates from outside inwards → edematous & congested
- If the condition is not treated within **8-12 hours** → becomes dark in color dark & finally gangrene.
- The spermatic cord shows the twists (one or more).
- If torsion persists, the blood vessels are thrombosed.
- The scrotal skin becomes red and edematous.



Clinical picture

1. Sudden severe agonizing pain in scrotum, groin & lower abdomen
2. Associated symptoms → vomiting (not persistent).
3. Scrotum is swollen with irreducible acutely tender swelling.
4. The overlying skin is edematous and red
- 5- The testis is higher than normal.
- 6- Shock with tachycardia and sweating may be present



Differential diagnosis

- 1-Acute epididymo-orchitis
- 2-Torsion of an undescended testis has to be differentiated from a strangulated inguinal hernia

	Torsion of testis	Acute epididymo-orchitis
Age	Usually adolescents & children	Usually adults or elderly
History	Sometimes mild trauma	Usually UTI symptoms
Temperature	Normal or slightly elevated	Elevated
Elevation of scrotum	Does not alleviate pain	Partial pain relief
Urgent urine analysis	Free	May show pus cells
Urgent Doppler or Duplex	Obstructed testicular vessels	Patent testicular vessels

Investigations

- 1- Urgent Duplex scan → detect blood flow obstruction in vessels
 - 2- Exploration
- Indication: Duplex scan is not available & diagnosis is suspected

- Exploration is indicated even if it proves negative
- Any delay may lead to loss of the testis.

Treatment

Urgent surgery

- Steps: 1- Scrotal incision 2- Undone torsion.
3- If the testis is viable, the tunica vaginalis is everted and the testis is fixed to the bottom of the scrotum to prevent recurrence.
4- If the testis is gangrenous → orchidectomy
5- The opposite testis should be fixed at the same time as the predisposing anomaly is commonly bilateral.

Inflammatory conditions of the testis and spermatic cord

Acute non-specific epididymo-orchitis

Incidence A fairly common disease

Mode of infection

- 1- Ascending infection (the usual route)
- Bacteria ascend through lumen of vas 2^{ry} to urethritis, prostatitis and seminal vesiculitis → spontaneously or after instrumentation (urethral catheterization or cystoscopy).
- **The commonest organisms:** gonococci, E. coli, proteus, streptococci, staphylococci & Chlamydia.
2- Blood borne infection (rare) 2^{ry} to specific fever as mumps.

Clinical picture

Symptoms

- General: 1- History of prostatitis or mumps.
2- Constitutional symptoms as fever, malaise & body aches
Local: Acute severe **scrotal pain** (the main symptom)

Signs

- 1- Epididymis & testis are swollen, tender with red & edematous skin
2- A small secondary hydrocele may be present.
- Inflammation may subside spontaneously (recurrence is common)
- Commonly pus cells are detected on urine analysis.

Treatment

- 1- An antibiotic for **2-3 weeks** (usually a member of quinolones)
2- Rest for the first few days

Chronic specific epididymo-orchitis

Filariasis

- 1- May follow an acute attack or is chronic from the start.
2- As the disease affects the lymphatics → spermatic cord is matted, (Vas cannot be differentiated from the other cord constituents)
3- There are firm to hard masses in the cord, but breaking down does not occur
4- There may be 2^{ry} hydrocele.

- Inflammation of the testis alone (**orchitis**) is rare. It is usually associated with inflammation of epididymis (**epididymo-orchitis**) or with spermatic cord & epididymis (**Funiculo-epididymo-orchitis**)
- It may be acute or chronic

Tuberculosis

1- The primary site of affection → the epididymis

2- Type of patient: mainly in young males.

3- Clinical picture:

a) Multiple tubercles affect the tail of epididymis → nodules enlarge

→ caseation & cold abscess formation → adherent to the skin and bursts → tuberculous sinus on the posterior aspect of scrotum

b) Vas deferens is thickened & studded with multiple tubercles (**beading**)

c) The testis usually remains free for a long time.

- Treatment: medical by anti-tuberculous drug, if no response → excision of the vas deferens and epididymis

Bilharziasis

- Affection of the cord or testis can occur as complication of urinary or intestinal disease.

- It is a chronic disease from the start.

- Site of affection: lower part of the cord and the epididymis.

- Clinical picture: 1- The cord is thickened but is not matted. It contains Bilharzial masses that vary greatly in size.

2- The testis and the tunica vaginalis are usually normal

Neoplasms of the testis

Incidence

- **99%** of testicular neoplasms are malignant
- They constitute **1-2%** of malignant tumours in males
- Occur at a relatively young age

Etiology

Incompletely descended testis (especially intra-abdominal variety)

- Increases incidence of malignancy **15 times** than normal population
- Surgery for undescended testis to bring it to the scrotum does not reduce the high susceptibility to malignancy

Classification

Germ cell tumours	Interstitial tumours (rare)	Lymphoma (rare)
1- Seminoma 40% 2- Non seminomatous germ cell tumours (teratomas) 32% 3- Combined seminoma and teratoma 14%	1- Leydig cell tumour 2- Sertoli cell tumour	

Seminoma

-Incidence: The commonest neoplasm of the testis.

- Type of patient: It occurs between **30-50 years** of age.

- Macroscopically: 1. the testis is large, firm and smooth.

2. Cut section is homogenous and pink creamy in color.

3. Sometimes, it is lobulated due to presence of fibrous septa.

4. Necrosis may occur in rapidly growing tumours.

- Histologically: 1. seminoma is derived from the seminiferous tubules → cells resemble spermatocytes (rounded or oval with clear cytoplasm and large rounded nuclei)

2. Cells are arranged in sheets separated by fibrous tissue stroma

-Spread: mainly by lymphatics and rarely by blood



Teratomas

-Type of patient: between the ages of **20-35 years**

-Origin: totipotent cells which can differentiate into the 3 embryonic layers (ectoderm, mesoderm and endoderm)

- Macroscopically: cut section is heterogeneous → show cysts that contain gelatinous material & areas of haemorrhage & necrosis.

- Histological classification:

a) **Differentiated teratoma** → no histologically recognizable malignant components but may metastasize → not considered benign (best example is dermoid cyst)

b) **Malignant teratoma intermediate** (commonest)

c) **Malignant teratoma anaplastica** → composed of undifferentiated embryonal cells of yolk sac origin → produce alpha-fetoprotein.

d) **Malignant teratoma trophoblastica** (the most aggressive)

This was previously known as choriocarcinoma

Interstitial cell tumours

- Onset: arises early in life either

- Origin:

a) **Cells of Leydig** (occur before puberty) → produce excessive amounts of androgens → infant Hercules (sexual precocity and extreme muscular development).

b) **Cells of Sertoli** (occur after puberty) → produce oestrogens → gynaecomastia, loss of libido and aspermia

Clinical picture

A) Typical cases

Symptoms

- Painless testis enlargement with sense of heaviness **1st symptom**

- Pain is present in **30%** of the cases.

Signs

- Testis is enlarged, hard, smooth & heavy. Later → soft bossy areas

- Testicular sensation is lost early in the course of the disease.

- In 10% of cases there is history of trauma that attracts the patient's attention to the presence of a swelling

Staging

Stage I:

Tumour in testis only

Stage II:

Involvement of LNs below the diaphragm

Stage III:

Involvement of LNs above diaphragm

Stage IV:

Systemic metastases

- Lax secondary hydrocele is present in **10%** of cases.
- The epididymis may be infiltrated with the tumour.
- The para-aortic LNs may be palpable just above the umbilicus.
- The inguinal LNs are not affected unless scrotal skin is infiltrated.
- The liver may be palpable.
- Symptoms due to lymphatic or blood metastases may be 1st presentation (as epigastric pain or cough, dyspnea & haemoptysis)
- The left supraclavicular nodes may be enlarged by metastases.
- Hormonal effects are present with interstitial cell tumors:
 - a) Infant Hercules
 - b) Feminising manifestations

B) Atypical presentations

- 1- Some cases may simulate epididymo-orchitis with acute pain and swelling that is due to haemorrhage.
- 2- Hurricane type → fatal termination due to metastases from highly malignant tumours.
- 3- Slowly growing type which takes **2-3 years** to develop.

Investigations

1- Tumor markers

a) **Beta fraction of human chorionic gonadotrophin**

- Raised in: i) 100% of patients with choriocarcinoma
ii) < 10% in seminoma
- Sustained elevation after treatment suggests residual disease.

b) **Alpha fetoprotein (AFP)**

- Elevated in non-seminomatous germ cell tumors (teratomas) but never in seminomas

c) **Lactic dehydrogenase**

2- Scrotal ultrasound

Confirm the presence of the testicular tumour & differentiate it from other lesions, e.g., epididymitis

3- Chest X-ray

4- Abdominal and chest CT scan

For lymph nodes, liver and pulmonary deposits

Treatment

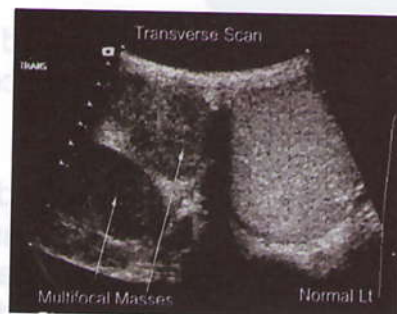
A) Initial treatment → high orchidectomy

-Operation:

1. Through inguinal incision, the spermatic cord is identified and isolated at the internal inguinal ring.
 2. A vascular clamp is applied as high as possible on the spermatic cord → avoid risk of blood dissemination while tumour manipulation
 3. The testis can now be delivered out and is inspected.
- If there is a frank tumour → excise the testis & the spermatic cord (after doubly ligating & dividing the cord latter at internal ring)
 - If in doubt → take biopsy for frozen section while clamp in place.

- An abdominal mass with an empty scrotal compartment should raise the suspicion of malignant transformation in an abdominal undescended testis.

- Biopsy should never be taken through the scrotum neither by incision nor by needle aspiration → opens a way for spread to inguinal LNs



A scrotal incision is contraindicated as it opens a way for spread to inguinal LNs

B) Further management (depends on type & stage of disease)

1- Seminoma

- Is radiosensitive → therefore used in early disease
- With advanced disease → more reliance on chemotherapy because of its systemic value.
- **Stage I** Radiotherapy for para-aortic lymph nodes.
- **Stage II** Radiotherapy that extends above the diaphragm
- **Stage III** Chemotherapy with or without radiotherapy
- **Stage IV** Chemotherapy

2-Teratoma

- The tumour is less sensitive to irradiation than a seminoma.
- **For stage I** → retroperitoneal lymphadenectomy or expectant policy
- **More advanced stages** → combination chemotherapy using cisplatin, etoposide and bleomycin.
- Follow up by tumor markers level, if:
 - a) Reduced to normal levels → success of therapy
 - b) Incomplete reduction → residual tumour.
 - c) Re- elevation → signifies recurrence

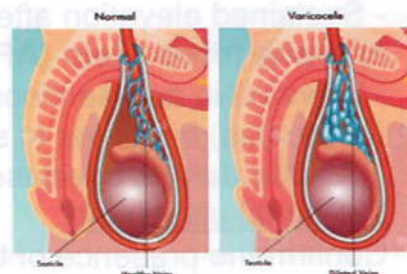
Varicocele

Definition

Elongation, dilatation and tortuosity of the pampiniform plexus of veins, i.e., varicose veins of the spermatic cord

Surgical anatomy

- 1- Veins of testis & epididymis form the pampiniform plexus
- 2- It is formed of 4-8 veins in the inguinal canal.
- 3- In the retroperitoneal area → reduced to **1-2** testicular veins.
- 4- The testicular vein ends in: a) IVC on the right side
b) Left renal vein on left side
- 5- The pampiniform plexus & testicular veins are devoid of valves except at their termination



Primary varicocele

Incidence Common condition

Etiology

- Its exact cause is not known but may be due to:
Congenital mesenchymal weakness → weakness of venous walls
(Associated with hernia, varicose veins, and flat feet)

- The left side almost always affects, the right side is rarely affected (sometimes it is bilateral)

- The tendency for the left side may be due to:

- 1- The right-angled termination of left spermatic vein into left renal vein blocks venous return (right vein terminations obliquely into IVC)
- 2- Lower position of left testis → longer & more dependent plexus.
- 3- Compression of the left testicular vein by the heavily loaded pelvic colon in constipated patients.

Clinical picture

Type of patient: Usually affects young adults (15-30 years)

Symptoms

- 1- Dragging sensation or aching pain in testis (especially on prolonged standing and in hot weather)
- 2- Infertility.

Signs

Inspection:

- The affected side of the scrotum hangs lower than normal.
- Large varicoceles are also visible
- Varicosities may be present in the scrotal skin

Palpation:

- The varicocele cannot be felt while patient is lying down. On standing → veins dilate → felt above testis like a **bag of worms**
- The varicocele forms a soft compressible inguinoscrotal swelling which gives a fluid thrill on cough.

Investigations

1- Doppler or duplex scan

Detect reversal of blood flow in the testicular vein.

2- Semen analysis (for cases of infertility)

3- Abdominal ultrasound

Exclude cases of 2^{ry} varicocele (those due to RCC)

Treatment

A) Conservative treatment

- In early cases → reassure the patient
- For pain, → scrotal suspender or close-fitting underpants (especially in hot weather)

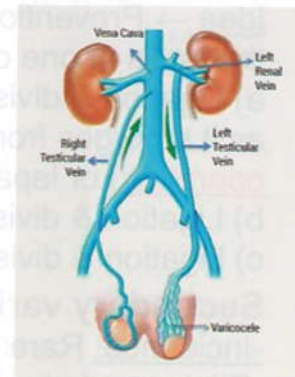
B) Surgical treatment

- Indications:

1-Infertility

- Due to defective spermatogenesis, Sperm count & viability
- Fertility improves in 30-50% of cases after surgery.

2- A large painful varicocele (when pain does not respond to conservative measures)



Complications

Hypofertility

particularly when bilateral → varicocele can lower sperm count & vitality → reduce fertility.

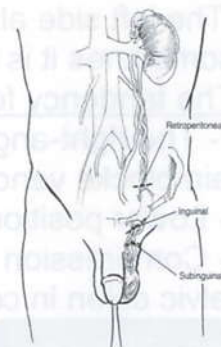
- Cause:

higher temperature in scrotum produced by venous congestion → impairs spermatogenesis

- Operations:

Idea → Prevention of venous reflux by attacking the venous return of the testis at one of these levels

- Ligation & division of testicular vein(s) in the extraperitoneal space as it emerges from the internal ring, either by open surgery (**Palomo operation**) or laparoscopically.
- Ligation & division of the pampiniform plexus in the inguinal canal.
- Ligation & division of pampiniform plexus at neck of scrotum



Secondary varicocele

-Incidence: Rare condition

-Etiology: obstruction of the venous flow in the spermatic vein by an abdominal tumour (usually RCC) → produced by:

- Intramural growth of the tumour in the renal vein or IVC
- Pressure from outside by the renal mass

- Differs from the primary type in:

- Occurs after the age of 40 years
- Affects both sides equally
- Develops rapidly and enlarges in a few weeks.
- Does not disappear on lying down or if the scrotum is elevated

-Treatment: is that of the cause

Epididymal cysts and spermatocele

Etiology Unknown

Pathology

- Arise from the head of the organ and filled with clear fluid
- Occasionally the cyst (called spermatocele) contains spermatozoa → make the fluid turbid

Clinical picture

Symptoms

- The patient complains of a painless scrotal swelling.
- The cyst is usually small (sometimes attains a large size → looks to the patient like a third testis)

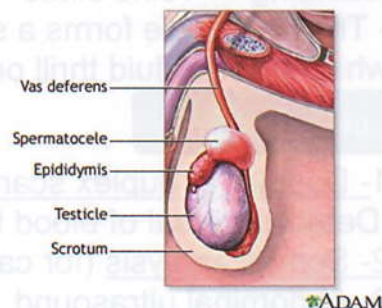
Signs

- On examination, the swelling is purely scrotal (lying just above & slightly behind the testis)
- Epididymal cysts are non-tender, cystic and translucent (with the exception of spermatoceles)

Treatment

- Most epididymal cysts deserve no treatment.
- Large or rapidly growing cysts → surgically excised.

Take care not to injure the epididymis while removing the cyst



• The main difference from a vaginal hydrocele is that an epididymal cyst is felt separate from the testis.



Hydrocele

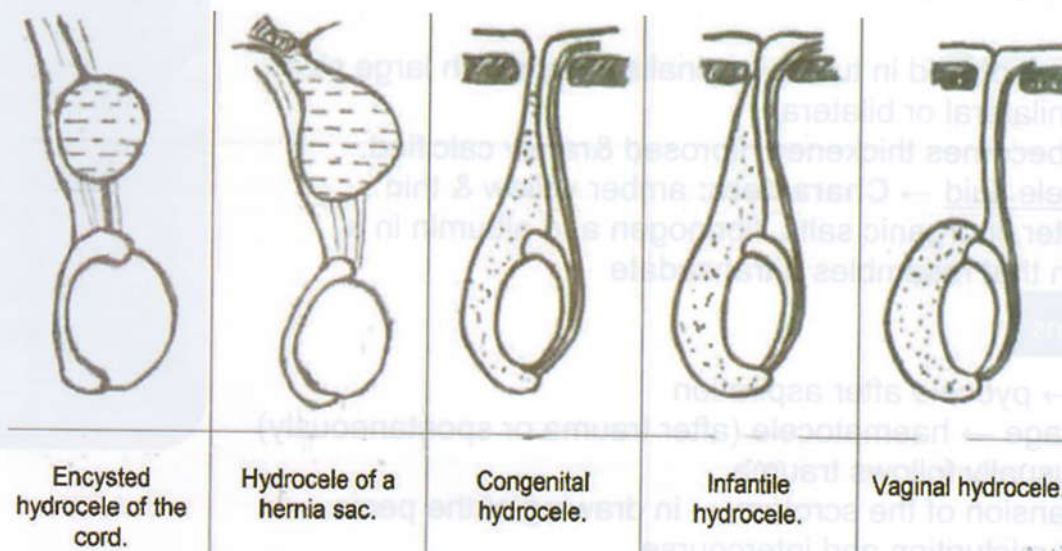
Definition

A collection of clear serous fluid inside a part of processus vaginalis

Classification

Hydrocele of the spermatic cord	Hydrocele of the tunica vaginalis
1- Encysted 2- Hydrocele of a hernia sac	1- Congenital 2- Infantile 3- Vaginal, primary or secondary

Hydrocele of the spermatic cord		
	Encysted hydrocele of spermatic cord	Hydrocele of a hernia sac
Etiology	-Persistence of the middle part of the processus vaginalis. - Although congenital, it usually appears in childhood as fluid takes time to accumulate	Fluid distension of an empty hernia sac which has been shut off from the peritoneal cavity by omentum or by adhesions
Clinical picture	A painless, small, translucent, cystic swelling at neck of the scrotum. - <u>Shape</u> : oval with the long axis along the cord. - <u>Mobility</u> : from side to side but not from above downwards. - The cyst is attached to testis by a fibrous band, representing obliterated portion of processus vaginalis. - With gentle traction on the testis, it is pulled downwards and becomes fixed.	1. There is history of a hernia. 2. Later, an irreducible pyriform translucent cystic swelling in the upper part of the cord. - <u>Mobility</u> : from side to side but not from above downwards. - Gentle traction on the testis does not change the position nor mobility of the swelling
Treatment	Excision.	Is that of the hernia



Hydrocele of the tunica vaginalis

Congenital hydrocele

- Etiology: Persistence of the whole processus vaginalis
- The communication with the peritoneal cavity is too small to permit the development of a hernia.
- C/P: 1. Cystic translucent, inguinoscrotal swelling.
- 2. The hydrocele is intermittent → fills gradually on standing & empties very slowly if the patient lies down & scrotum is elevated.
- Treatment: is **surgical**.
- Through an inguinal incision, the sac is divided into two parts:
 - a) The upper part → transfixed at level of internal ring as a hernia
 - b) The lower part → either everted or is just left undisturbed.

Infantile hydrocele

- Etiology: incomplete obliteration of the processus vaginalis → the tunica extends up to the internal ring, but does not communicate with the peritoneal cavity.
- The differentiation from a congenital hydrocele is only at operation.
- Treatment: by eversion of the tunica.

Vaginal hydrocele

Definition A fluid collection in the tunica vaginalis

Etiology

- 1- Idiopathic (primary hydrocele)
- 2- Due to an underlying lesion (secondary hydrocele)

Primary vaginal hydrocele (the most frequent type)

Etiology

- The cause of this condition is not known.
- Possible causes: 1-Diminished fluid absorption by tunica vaginalis
- 2-Chronic congestion & irritation by repeated trauma

Pathology

- Accumulation of fluid in tunica vaginalis (may reach large size)
- It may be unilateral or bilateral
- The tunica becomes thickened, fibrosed & rarely calcified.
- The hydrocele fluid → **Characters**: amber yellow & thin

Content: water, inorganic salts, fibrinogen and albumin in a concentration that resembles a transudate

Complications

- 1- Infection → pyocele after aspiration
- 2- Haemorrhage → haematocele (after trauma or spontaneously)
- 3- Rupture, usually follows trauma.
- 4- Huge expansion of the scrotum → in drawing of the penis → interfere with micturition and intercourse

Secondary hydrocele

Incidence:

uncommon.

- Etiology: 2nd to a disease of the cord or the testis.

- This disease may be acute (epididymo-orchitis) or chronic (malignant or chronic Inflammation)

- C/P: usually small and soft.

- With testicular cancer, the fluid may be Bloodstained

- Treatment: is that of the cause.

Clinical picture

Type of patient: middle aged and elderly males.

Symptoms

Painless scrotal swelling

Signs

Inspection: pyriform swelling

Palpation:

1- Purely scrotal swelling

(Fingers can reach above the swelling at the neck of the scrotum)

2- The swelling is cystic with smooth surface & no impulse on cough

3- The hydrocele fluid surrounds the testis, which is often impalpable

Transillumination → translucent



Treatment

Essentially surgical

- Methods:

1- Eversion of the tunica vaginalis

- A scrotal incision is done, tunica vaginalis is opened & evacuated.

- The cut edges are everted behind the testis & sutured behind epididymis by continuous sutures

2- Excision of the tunica vaginalis

- Indication: a) large hydrocele

b) Thick walled tunica vaginalis.

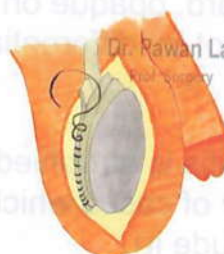
3- Lord's operation

- Indication: suitable for cases where the tunica is not thickened.

- A small scrotal incision is made through all layers including tunica.

- The testis is allowed to prolapse through the wound.

- The tunica is plicated by a series of radial sutures around the testis.



- The spermatic cord, epididymis & testis should be examined to exclude cases of 2nd hydrocele
- Scrotal ultrasound helps examination of a testis that is difficult to palpate

- Aspiration is not recommended because:

1. Recurrence is certain
2. May be complicated by infection or haemorrhage



Haematocele

Definition

Accumulation of blood or blood-stained fluid in the tunica vaginalis

Acute haematocele

Etiology

1- Trauma to the testis, injuring the tunica albuginea

2- Postoperative, e.g., testicular biopsy.

3- Aspiration of hydrocele

4- Torsion of the testis

5- Acute funiculo-epididymo-orchitis.

Clinical picture

Symptoms: acutely painful swelling of the scrotum.

Signs: swelling is tense, tender & opaque with transillumination

Treatment

Surgical evacuation of the blood and excision of the tunica vaginalis

- If there is a tear in the tunica albuginea → repair it

Chronic haematocele (old clotted haematocele)

Blood clots → Fibrosis & thickening of the tunica

Etiology

- 1- Neglected acute haematocele
- 2- Repeated minor hemorrhages in blood diseases.

Complications

- 1- Infection.
 - 2- Compression & atrophy of the testis
 - 3- Calcification
- (In old-standing cases & may be mistaken for malignancy)

Clinical picture

Symptoms: Heaviness & swelling of the scrotum

Signs: swelling is hard, opaque on transillumination and the contents of the scrotum cannot be differentiated

Treatment

Orchidectomy (testis is atrophied anyway)

Indications: majority of cases which are late & confused with malignancy (to exclude it)

Pyocele

Definition Collection of pus in the tunica vaginalis

Etiology

- 1- Infected haematocele.
- 2- Infected hydrocele especially after tapping.
- 3- Secondary to suppurative funiculo-epididymo-orchitis

Clinical picture

- **General:** constitutional manifestations.

- **Local:** pain, redness, hotness, edema and tenderness.

Treatment Incision and drainage of pus

Chylocele

Definition

Chyle (lymph) accumulates in the tunica due to rupture of lymphatic vessels in case of Filariasis

Clinical picture

Swelling which is similar to hydrocele but trans-opaque

Phimosis

- **Definition:** a congenital disorder where there is narrowing of the preputial orifice and inability of the prepuce to retract beyond the glans penis

- **C/P:** the prepuce balloons during micturition.

- **Complications**

Trial to forcibly retract the prepuce → paraphimosis & strangulation of the glans

- **Treatment:**

1. Many cases resolve spontaneously in childhood
2. Persistent cases → circumcision



Clinical diagnosis of a scrotal swelling

Q1. Can the examiner's fingers meet above the swelling?

1) If cannot → it is an inguinoscrotal swelling → oblique inguinal hernia that reaches down to the scrotum or a varicocele.

- To differentiate:

a) Hernia → expansile impulse on cough & usually reducible (may be accompanied by a gurgling sound)

b) Varicocele → feels like a bag of worms, compressible, disappears on lying down and produces a palpable thrill on cough.

2) If can get above the swelling → a purely scrotal swelling.

Q2. What is the consistency of the purely scrotal swelling?

1) **Cystic** → vaginal hydrocele, epididymal cyst, encysted hydrocele of the spermatic cord or hydrocele of a hernia sac.

2) **Solid** → testicular cancer, old clotted haematocoele, chronic epididymitis (tuberculous or non-specific)

Q3. In case of a cystic scrotal swelling is testis felt separate from the swelling or impalpable because it lies within swelling?

1) **Impalpable** → vaginal hydrocele and confirm by transillumination

2) **Felt separate from the swelling**

a) Epididymal cyst → low in scrotum & is attached to epididymis

- It cannot be moved separate from the testis.

b) Encysted hydrocele of the cord → moved separate from the testis.

- Downward traction on the testis → limits swelling mobility

c) Hydrocele of a hernia sac → traction on testis not affect mobility

Q4. In case of a hard-scrotal swelling, does it include the testis, or is it confined to the epididymis?

1) Includes the testis → testicular cancer or old clotted haematocoele

2) Confined to the epididymis → tuberculous and chronic non-specific epididymitis

Important points

- A retractile testis is a normal finding in infants & young children → requires no treatment

- An undescended testis should be operated upon before **2 years** of age

- Testicular torsion is a surgical emergency if in doubt, urgent surgery is still recommended in order to save the testis.

- Never puncture nor incise the scrotum in a case that is suspected to be testicular cancer → opens a way for spread to inguinal LNs

Burns and principles of plastic surgeries

Skin histology

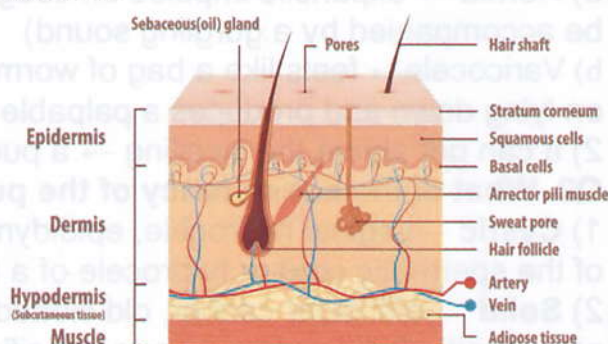
- Formed of 2 layers:

1- Epidermis: formed of stratified squamous epithelium

2- Dermis: formed of connective tissue.

- The skin appendages (hair follicles, sweat glands & sebaceous glands) arise from the epidermis & grow down into the dermis.

- When epidermis is damaged in burns, epithelium from these appendages can creep up to cover the denuded area.



Functions of skin

1. Forms a barrier against invasion by micro-organisms.
2. Prevents excessive loss of water from the body.
3. Regulates body temperature by secretion of sweat.
4. Sensory receptors in skin protect the body from injurious insults.
5. Formation of vitamin D.

Etiology of burn

A) Thermal injuries include:

1- Scalds: caused by boiled liquids (often affect children)

2- Flame burns: affect any age & often due to setting cloth on fire (the most common burn in industrial accidents)

3- Steam burn: Inhalation & upper or lower respiratory airway burns due to exposure to hot gases

B) Electric burns

- Usually due to contact with an electric socket or low voltage battery

- Contact with high voltage may be fatal since blood & bones are good conductors → every organ in the body may be affected.

C) Chemical burns are due to either acids or alkalis.

- Skin is the 2nd largest organ in body (by size & weight) next only to muscles
 - Loss of large amount of skin, if not replaced, is incompatible with life.
 - Skin has limited ability of regeneration & lacks much of functional reserves of internal organs

Pathology of burn

1. The extent

- Definition: the percent of burnt skin surface area in relation to the whole body surface area.

- This follows the rule of 9

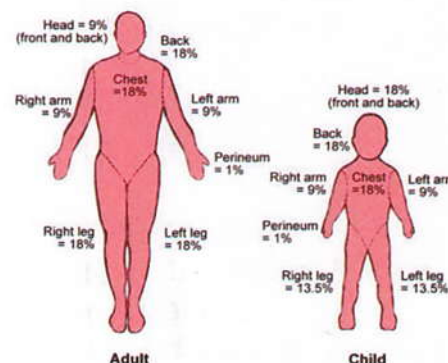
- In children the rule of 9 needs some modification due to large size of head in comparison to the rest of body.

- Classification:

1- Major burn → > 30% of the body surface area

2- Intermediate burn → 15-30% in adults & 10-30% in children

3- Minor burn → <15% in adults and 10% in children



2. Depth

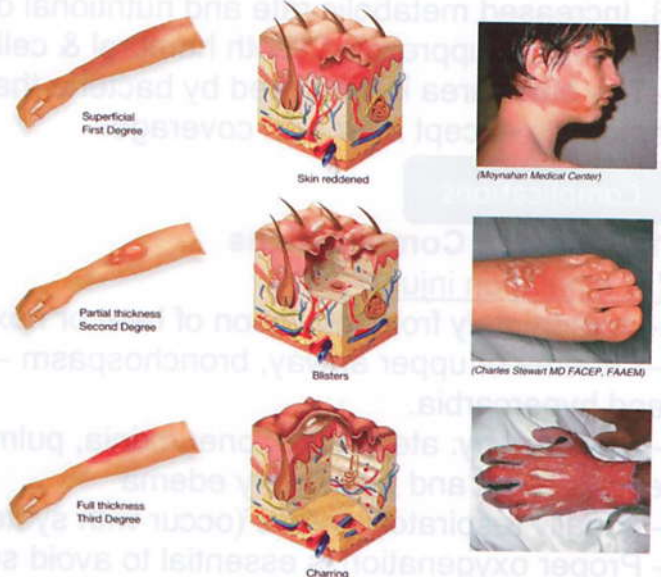
	1 st degree burns	2 nd degree burns	3 rd degree burns
Damage	Only the epidermis → skin erythema - The usual example is sunburns	The epidermis and a portion of the dermis	The epidermis & dermis are completely destroyed
Healing	Heal rapidly	- Provided that no infection → epithelial regeneration can occur from remnants of hair follicles & sweat glands in dermis & heal in 3 weeks - If infection occurs → epithelial elements are destroyed and will be changed to a full thickness burn - Should heal within 3 weeks	Epithelial coverage only by epithelial migration from the edges of the burnt area (slow process) After eschar separation by the 3 rd week → Prepare for skin grafting
Appearance	- Formation of blisters surrounded by erythema. - Their surface is mottled red & usually moist due to plasma exudation		- The area is dry - White or black eschar (burn slough) present with possible visible thrombosed S.C. vessels
Presence of pain	Very painful and sensitive to air		Painless (due to loss of the terminal nerve endings) → elicited clinically by the pinprick test

- 2nd degree burns further subdivided into:

i. Superficial partial thickness burns Heal with epithelialization in 10- 14 days

ii. Deep partial thickness burns
- Slower to heal (25-35 days)
- Cells from the residual uninjured epithelium of deep sweat glands and hair follicles creeps up to cover the surface

-N.B: Burn can be classified into
1. Superficial → 1st & 2nd degree
2. Deep → 3rd degree



Histopathology

- Human skin is injured by heat in two ways:

- 1- An immediate direct cellular injury
- 2- A delayed injury due to progressive dermal ischaemia

- The degree of tissue destruction is related:

- 1- Temperature
- 2- Duration of heat exposure

- Cause of cell damage is protein denaturation & many are reversible. However, at temperatures $> 45^{\circ}\text{C}$ → irreversible

- Histologically, skin thermal injury results in 3 distinct trauma zones:

1-The central inner zone (zone of coagulation)

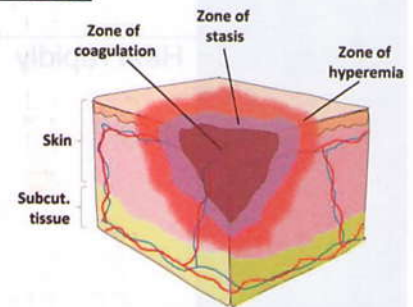
- Forms the inner layer of the visible burn eschar.
- If left, the eschar separates within 3 weeks leaving either:
 - a) Bed of granulation tissue (in full thickness burns)
 - b) Re-epithelialized bed (in partial thickness burns).

2- The intermediate zone (zone of stasis)

- Surrounds the zone of coagulation.
- It contains viable tissues that may die over the next 48 hours if tissue oxygenation and adequate nutrition are not maintained.

3- The outer zone (zone of hyperaemia)

- This area contains inflammatory mediators (prostaglandins, histamine and kinins) → formation of tissue edema.
- Normally recover within 7-10 days unless subjected to infection.



Pathophysiology

The following pathological sequelae occur in the burnt patient:

1. Increased capillary permeability → loss of fluids & proteins in the damaged area (maximum in the 1st 8 hours & continues for 48 hours)
2. Excessive loss of water by evaporation through the burnt skin
 - A patient with a full thickness burn can lose up to **200 ml/hour**.
 - The body loses **560 KCal** for every liter of water evaporated → severe dehydration & catabolism.
3. Increased metabolic rate and nutritional demand.
4. Immunosuppression (Both humoral & cell-mediated)
 - The burnt area is colonized by bacteria that are impossible to eradicate except after skin coverage.

Complications

1. Systemic Complications

(a) Inhalation injury.

- i- Immediately from inhalation of heat or noxious gases → asphyxia → edema of upper airway, bronchospasm → progressive hypoxia and hypercarbia.
- Followed by: atelectasis, pneumonia, pulmonary emboli, emphysema and pulmonary edema
- Finally respiratory failure (occur with systemic sepsis)
- Proper oxygenation is essential to avoid such complications

The rate of burn healing depends on the density of the surviving adnexal epithelial structures within dermis (especially hair follicles & sweat glands) which are present deep to epidermis.

- A) In a partial thickness burn** → healing achieved by epithelialization within **3 weeks** without any surgical intervention
- b) In a full thickness burn** → eschar separation occurs at about **4 weeks** → clear granulation tissues → heals by fibrosis and scarring (If left without grafting)

(b) Neurogenic shock: caused by pain.

(c) Hypovolaemic shock: if fluid losses are not properly corrected

(d) Renal failure.

- Acute RF can occur after prolonged uncorrected hypovolaemia

(e) Gastro-intestinal complications.

i- **Curling's ulcer** (acute ulceration of stomach & duodenum)

Rarely seen nowadays with routine prophylactic use of antacids & H₂ receptor antagonists during the initial burn management

ii- **Ileus** usually occurs early in post-burn period and may necessitate the insertion of a nasogastric tube.

iii- **Acute colon ulcerations**: may be deep reaching the serosa (Secondary to fungal infection)

(f) Multiple organ failure (Poor outcome)

- May follow major burns particularly with sepsis

2. Local Complications

(a) Early local complications

I- Infection

- The primary cause of death in burnt patients.

- May be bacterial, viral or fungal

- Can occur from both exogenous and endogenous sources

- Complications: lead to septicaemia and septic shock.

- Timing: usually between **4-7 days** post burn.

- Treatment: by prevention through proper local burn wound care (The value of systemic antibiotics in prevention is doubtful)

II- Constricting eschars

- In deep circumferential burns of the limbs and chest → restrict chest expansion

- Treatment: by urgent escharotomy

III- Suffocation

- Due to edema in burns of the face & neck

- Treatment: urgent tracheostomy

IV- Compartment syndrome

(b) Delayed local complications

1- Contractures across joints

2- Scar formation (**hypertrophic or keloid**).

3- Malignant transformation (**Marjolin's ulcer**) in long-standing unstable scars is rare.



A) First aid

1. Assure a patent airway

-If there is airway obstruction → endotracheal intubation

2. A strong analgesic as 50 mg pethidine is **administered IV**

3. Tetanus prophylaxis.

4. Pour Saline or tap water (**at room temperature**) over burnt area for **15 minutes** → limit burn depth, decrease edema & relieve pain

B) Admission to hospital

- Indications:

1. Inhalation injury.

2. Burn size >**15%** in adults or **10%** in children

3. Any full thickness burn.

4. Burn in association with trauma or co-morbidity.

5. Electric burns

6. Chemical burns

- Resuscitation:

1- Inserted a wide bore IV. Cannula rapidly before veins collapses

2- Introduce a Foley urethral catheter to check urine output.

C) Resuscitative fluid therapy

- The amount and rate of fluid replacement are determined by:

1- The patient's weight

2- The percentage of the total body surface area injured.

- Given for the first **48 hours** and after that according to the response of the patient a maintenance therapy may continue.

- The greatest fluids loss occurs during the first **8-12 hours** post-burn and continues more slowly over the next **12-16 hours**.

- The amount infused during the first 24 hours is **2 mL/percent** surface area burn/kg body weight.

- $\frac{1}{2}$ the amount calculated is given over the first **8 hours** and the other $\frac{1}{2}$ over the next **16 hours**.

- Also half the calculated amount is given over the 2nd day.

- Concerning the types of fluids, it varies in each formula where

- In all formulas the daily caloric needs should be provided by the administration of **2000 ml** of glucose **5%** solution.

- Administration of blood is needed in major deep burns (started after **48 hours**, guided by the haematocrit value)

- The commonest formulae used is: **Parkland's formula**

- (**4 ml/kg/% burn lactated Ringer's solution/day**)

- $\frac{1}{2}$ the amount is given in the 1st 8 hours, $\frac{1}{4}$ in the 2nd 8 hours & $\frac{1}{4}$ in the 3rd 8 hours.

- In all formulae, the maximum percentage of burn calculated is **50%**, otherwise serious over infusion may occur.

- Oral intake is avoided during the 1st 8 hours to avoid gastrointestinal complications and is started gradually after that.

- IM injections are avoided as absorption is poor.

- Using ice cold water from a refrigerator is **contraindicated**

→ induce more tissue damage

- Minor burns (< **15%** in adults & **10%** in children)

are treated as outpatients by dressing using the proper local chemo-therapeutic & analgesia

- Treatment essentially consists of:

1. Fluid therapy → compensate for losses

2. Local burn wound care

- The types of fluids used in replacement varies in each formula:

1. Only crystalloids (**saline or lactated Ringer's**) over 1st day,

2. Others → $\frac{1}{2}$ the amount should be crystalloid & other $\frac{1}{2}$ to be colloids (Dextran)

- The value of systemic antibiotics in infection prevention is controversial

- Monitoring the adequacy of IV resuscitation by:

1. Regular check-up of vital signs.
2. Urine output should be **0.5-1 ml/kg/hour**.
3. C.V.P. in critical cases.

D) The nutritional status

- Should not be neglected.
- Patients with extensive burns are liable to have a serious catabolic status due to: 1- Anorexia 2- Extensive water & caloric losses 3-Sepsis (if present)
- I.V. hyperalimentation has made it easy to correct this problem. However, enteral nutrition is preferred.

Local burn wound care

A) Early care

1. Escharotomy

- Do it immediately for constricting eschars (in limbs & chest)
- Sometimes, fasciotomy (in deeper burns) may be limb saving

2. Cleansing

- Remove loose skin & initial conservative debridement to avoid infection.

3. Topical antimicrobial agents

- The ideal topical cream should be: in a water soluble base, prevents dryness, not painful, non-allergenic, non toxic & most importantly bactericidal but not injurious to viable cells in the burn wound

- The 3 most commonly used are: i) Silver sulphadiazine
ii) Silver nitrate solution iii) Mafenide acetate

4. After local cream application, the wound is managed by either:



	Exposure method	Closed method
Definition	Leave the wound exposed (requires patient isolation in completely aseptic atmosphere & following all aseptic rules by the personnel dealing with patient)	Cover the wound by a bulky occlusive dressing that is changed every 2- 3 days depending on the state of the wound.
Indication	1- Burns of the face, neck & perineum 2- Burns involving one side of the trunk	1. Circumferential burns 2. Limbs (to prevent stiffness & adhesions)
Advantages	1. More comfortable to the patient. 2. Avoids repeated change of dressings (burden & expensive) 3. Inhibits the growth of anaerobic bacteria	1. ↓ cross infection 2. ↓ fluid loss by evaporation 3. ↓ pain by covering exposed nerves 4. ↓ tissue edema by compression

B) Later care

For deep burns:

1. Autologous skin grafting

- **Thiersch grafts** (Partial thickness skin grafts) are commonly used to cover the large raw areas produced by burns.

- A graft is applied after either:

I) Spontaneous separation of eschar (**3-4 weeks**)

II) Early burn wound excision → the preferred method for deep burns of the hand.

2. Biological dressings

- Indications: 1- Autografts are not enough

2- Local wound conditions are not favourable

- Advantages:

1- The wound will be less painful

2- Minimize fluid and protein

3- Control infection

- Examples:

1- Allograft (cadaver's skin)

2- Xenograft skin (pig's skin)

3- Amniotic membrane.

4- More recently, artificial skin substitutes are used

- Applied only after eschar removal & changed every **3-4 days**.

Prevention of contractures by

1- Proper splintage

2- Immobilization in the best functioning position

3- Physiotherapy.

Following early wound care, all partial thickness burns heal within **2-3 weeks** while full thickness burns require closure by autogenous grafts

- Grafts are applied **3-5 days** post-burn (before bacterial infection sets in) after tangential excision of the damaged dermis & reaching healthy bleeding tissues

- Biological dressings are only temporary and permanent closure can be achieved only by autologous skin graft

Special types of burns

A) Electric burns

- Divided into high & low tension injuries according to the voltage responsible for the injury (whether above or below **1000 volts**).

- The tissue damage is due to the passage of the electric current through blood and bones.

- The affected blood vessels usually show late thrombosis → delayed progressive ischaemic necrosis

- Clinically 3 types of skin damage may result:

1. Contact burns occur at points of current entry & exit from body

2. Burns from current exit and re-entry at adjacent parts.

3. Thermal burns from ignition of clothing due to the heat generated by the current passing through the body



B) Inhalation burns

- Flames or very hot air may be inhaled causing burns in:

- 1- Upper respiratory tract → laryngeal edema → stridor
- 2- Lower respiratory tract → pneumonia & ARDS → RF

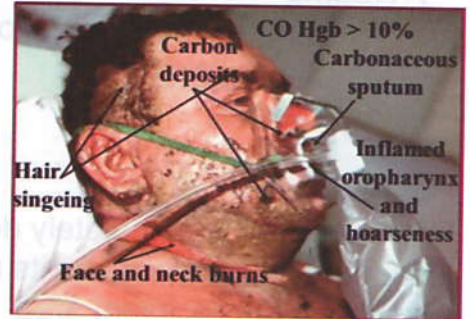
- Suspected in:

- 1- Closed space burns
- 2- Facial burns
- 3- Burnt nasal hairs
- 4- Carbonaceous sputum

5- Wheezing, pharyngeal edema or hoarseness of voice.

- Management:

- 1- Early intubation
- 2- High-flow oxygen
- 3- Hospital admission with possible need for mechanical ventilation



C) Chemical burns

- Chemical agents burn by oxidation, reduction, corrosion or protoplasmic poisoning

- The severity is determined by:

- 1- The strength of the agent
- 2- Its amount
- 3- Duration of skin contact and its mechanism of action.

- Management:

1- Systemic → by administering the proper antidote in case the agent is absorbed.

2- Locally → thoroughly clean the agent, all saturated clothes are removed and affected areas irrigated with huge amounts of water.

D) Cold injury

- Is a form of thermal injury that is due to exposure to cold

- Depends upon: 1- Duration of exposure 2- Temperature

- Usually affects hands, feet, ears and nose.

- Examples: frostbite, trench foot and chilblain.



Prognosis of burns

A) Burn factors

1. Extent (the most important factor)

- Mortality is about 50% if the extent of burn is 50%.

2. Depth. Deeper burns carry higher mortality & more disfigurement

3. Site. Burns of the face are the worst because they are likely to be accompanied by inhalation burns of the respiratory tract.

4. Infection. Septicaemia is the major killer of burn victims.

5. Type.

- High-voltage electric burns are the worst → usually fatal.

- Scalds with boiling water → superficial burns & the least harmful

6. Associated injuries

B) Patient factors

1. Age → extremes of age (children & elderly) → bad prognosis

2. Concomitant diseases as DM & coronary heart disease.

C) Treatment factors

- Those treated in specialized centres → better chance of survival and avoidance of complications.

Principles of skin coverage

- Skin defects result from:

- 1-Trauma
- 2- Burns
- 3-Following surgical resection of different benign & malignant lesions

- Methods of coverage:

A) Skin grafts

Definition

A tissue that is completely detached from its blood supply in the donor area and receives its new blood supply from the base of the wound in the recipient area

Types

	Split thickness (Thiersch) graft	Full thickness (Wolfe) graft
Definition	Includes epidermis & part of dermis	Includes epidermis & full thickness of the dermis.
Advantages	1. Survive easily (good take) 2. Donor site heals spontaneously	- <u>All qualities of normal skin:</u> 1.Minimal contraction 2.Resistant to trauma 3. Better sensation 4.Aesthetically better
Disadvantages	- <u>Few qualities of normal skin:</u> 1. Marked contraction 2. Weak resistance to trauma 3. Poor sensation 4. Aesthetically poor	1. Less take 2. Donor site must be closed surgically 3. Donor sites are limited

Graft survival

- A graft must obtain a blood supply from its recipient bed to survive
→Thus vascular status of bed determines skin graft survival (take)
- Neovascularization (new vessels that grow from the recipient bed) takes about **5-7 days** to be established depending upon;
 - 1- Status of the recipient bed
 - 2- Graft thickness
 - 3- Size of the graft
- During neovascularization, the graft needs to be supported by some form of immobilization and mild compression

Technique

I) Full thickness grafts

- Obtained from anywhere in the body using the scalpel blade.
- Taken from an area close to recipient site for better color matching, e.g. from above clavicle or post auricular area to cover facial defects

Skin graft may include an underlying tissue as S.C. tissue, muscle, Cartilage or bone→ termed a composite graft

Poorly vascularized tissues such as bare cortical bone (devoid of periosteum), bare tendon & bare cartilage will not take a skin graft but can be covered by flaps
-The thinner the graft the faster the take
- A small graft takes rapidly

II) Split thickness grafts

- Obtained by special knife (**dermatome**) from hidden areas as thighs
- The donor site heals with complete epithelization within **3 weeks**.
- The epithelium is derived from that of the skin appendages (hair follicles, sweat glands & sebaceous glands) that have deep extensions into the dermis.

B) Flaps

Definition

Tissues to be transferred from one site of the body to another with maintaining their blood supply for nourishment.

Type

1. Skin flaps

- Classified into:

a. Local flaps

i) **Random pattern flaps** → have no anatomically recognized blood supply → thus should have a strict length to width ratio to survive.

- A safe random pattern flap has a length to width ratio of **2:1**

ii) **Axial pattern flaps** → supplied by a known artery and have no limitations as regards length to width ratio.

- The whole area supplied by the specific cutaneous artery can be transferred safely.
- Adjacent nerves may be included → the flap will be a sensory one.

- May retain their skin attachment to donor area → **pedicled flaps**

- If they retain their blood supply without skin attachment (have a wider arc of rotation) → **Island flaps**

b. Distant pedicle flaps

- Keep the recipient attached to the donor site for a period of **2-3 weeks** to allow for neovascularization before separating the base

- Examples: i- flaps from abdomen or groin to resurface the hand

- ii- From one leg to another (**cross leg flap**)

2. Musculocutaneous (myocutaneous) flaps.

- These are axial flaps that receive blood supply from the underlying muscles through perforator vessels.

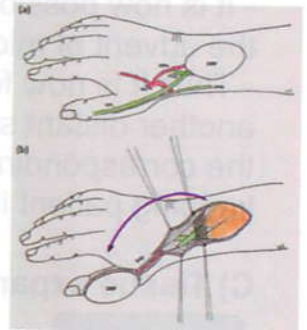
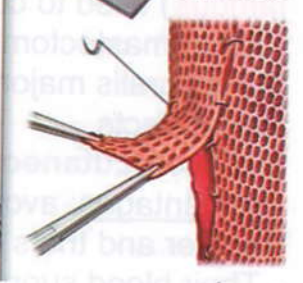
- The muscle is lifted from its bed with the overlying fascia and an area of skin (retaining the neurovascular pedicle of the muscle) then rotate the whole flap in an arc to cover the raw area

- The incorporation of the pedicled muscle in the flap enhances the survival of the cutaneous component.

Graft taken from patient's healthy skin



Skin is meshed to cover a large wound



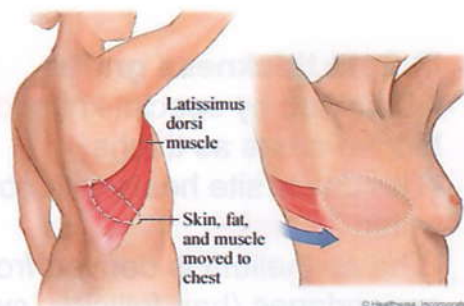
- Examples:** i- Latissimus dorsi myocutaneous flap (is famous) used to cover defects resulting from wide radical mastectomies
 ii- Pectoralis major myocutaneous flap to cover neck or face defects

3- Fasciocutaneous flaps

- **Advantages:** avoid the functional deficit of muscle transfer and the excess bulk of myocutaneous flaps
 - Their blood supply is through fasciocutaneous perforators.

4. Microvascular free flaps

- It is now possible to anastomose vessels $< 1 \text{ mm}$ in diameter with the advent of microvascular techniques.,
 - Thus it is now feasible to transfer an axial flap from one site to another distant site by anastomosing the artery & veins of the flap to the corresponding vessels in the recipient area without need for keeping patient immobilized for 2-3 weeks & possible joint stiffness



C) Tissue expanders

Definition

Are inflatable silicone implants.

Technique

- 1- They are placed subcutaneously in collapsed state.
- 2- Over several weeks the expander is gradually inflated with saline through a subcutaneous port.
- 3- The overlying skin is gradually stretched to accommodate a larger area.
- 4- Finally, the expanded skin can be used to cover defects and the tissue expander is removed



Reconstruction ladder



Aesthetic surgery

Definition

Surgery intended to improve appearance for correction of any disfigurement as in scar revision and in enhancement of the normal appearance by correcting a deformed nose or correcting the effects of the aging process in the face.

Examples

1- Rhinoplasty: Correction of a deformed nose.

2- Face lifting:

- **Aim**: get rid of the redundant excess skin of the face that occurs with aging.
- Done through scars that are hidden in hair or just in front of the ears in a natural crease.
- Recently can be carried out through endoscopic techniques.

3- Eyelids surgery:

- **Aim**: correction of the aging process affecting the eyelids through very fine incisions in their creases.

4- Breast surgery → either:

- a) Augmentation mammoplasty → to enlarge small breast by the insertion of a silicon implant under the breast.
- b) Reduction mammoplasty → to reduce large breast

5- Liposuction:

- **Definition**: body sculpturing surgery by aspiration of excess fat accumulating in the abdomen, buttocks or trochanteric areas.

- **Method**:

1- Insertion of a special cannula through **1cm** skin incision

2- The cannula is connected to a special machine with a high vacuum pressure to allow aspiration of the excess fat

The selection of patients for aesthetic surgery is important, since there is no pathology to correct but rather an improvement in the body image is asked for.

Sometimes the aspirated fat can be treated in a special manner & reinjected to augment other areas in the body with depressions (autologous fat injection)

Skin and subcutaneous tissue

Benign lesions

Sebaceous (epidermoid) cyst

Definition

A retention cyst caused by blockage of a sebaceous gland duct.

Pathology

- **Lining:** stratified squamous epithelium
- **Content:** a foul smelling, white material composed of keratin, epithelial cells and granular debris

Clinical picture

Type of patient: rarely before adolescence.

Symptom → Slowly growing cysts

- **Site:** most commonly in scalp, face, neck or scrotum but can occur anywhere except palm or sole of foot (devoid of sebaceous glands)

Signs

1. A small, well defined, cystic swelling which is usually attached to the skin at one point which is the site of the punctum
2. The swelling is mobile over the deep structures.
3. Sometimes a sebaceous cyst attains a large size.
4. The lesion may be solitary or multiple

Complications

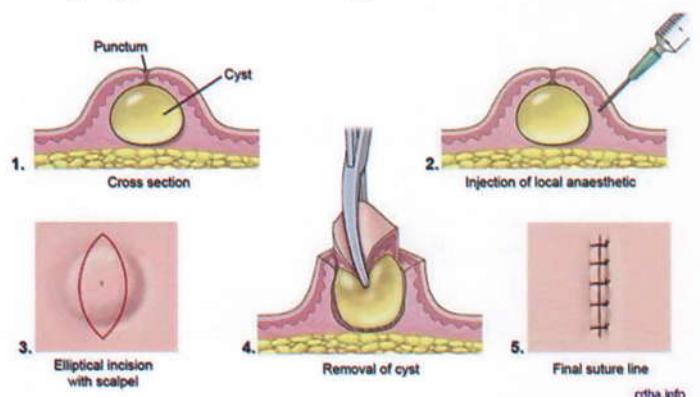
1- Infection (the commonest)

- **C/P:** 1. The cyst becomes painful and tender.
- 2. There is overlying redness and an abscess may form.
- Early cases → may subside by antibiotics.
- If an abscess forms → incised for drainage (excision is delayed later as infection makes cyst wall adherent to surrounding tissues)

2- Localized alopecia

Treatment

- Complete cyst excision with an ellipse of overlying skin containing the punctum to avoid recurrence
- If the cyst is small → local anaesthesia



Dermoid cyst

Definition

A cyst lined by stratified squamous epithelium & contains sebaceous material

Types

1- Sequestration dermoid cysts

- Etiology: subcutaneous inclusion of portions of surface epithelium along the lines of fusion of cutaneous dermatomes during fetal life.

- Time of presentation: after a few years when the cyst begins to distend (although present since birth)

- Sites: 1. External angular at the outer angle of the eye
2. At root of nose 3. Around the ear
4. along the midline of the body

- Clinical picture:

1. A welldefined, globular, cystic swelling not attached to skin
2. The underlying bone may be hollowed out & there may even be a connection to dura matter

2- Tubulo- dermoid cysts

- Etiology: distension of remnants of embryonic ducts such as:

- a) Thyroglossal duct → thyroglossal cyst
- b) Cervical sinus → branchial cyst

3- Teratomatous dermoid cysts (benign forms of teratomas)

- Lining: squamous epithelium

- Contents: teeth, hairs, bone, cartilage or glands.

- Site: 1. the ovary (mostly)
2. The testis 3. Posterior mediastinum

4- Implantation dermoid cysts

- Etiology: 2^{ry} to punctured wounds → displace some epithelial cells into S.C. tissue → retain their viability & form a dermoid cyst

- Site: 1. Fingers (mainly) 2. Palm & sole

- C/P: They are usually small and tense



Treatment

Surgical excision

- In children with dermoid cyst in the scalp → wait until closure of the skull sutures (as some cysts may communicate with the dura)

Papilloma

- Definition: a benign tumour usually pigmented which commonly develops in large numbers in the face, arms and upper trunk

- Etiology: may be a familial with autosomal dominant inheritance

- Pathology: composed of immature epidermal cells nourished by elongated dermal papillae.

- Course: Malignant transformation is rare.

- Treatment: Excision, cryosurgery or cautery



Callosity

-Definition: an area of superficial thickening of the skin due to continuous friction

-Site: 1. Mostly on toes & sole from wearing ill -fitting shoes
2. Fingers & hands of manual workers.

- Clinical picture:

1. Not painful & forms a circumscribed yellowish white plaque
2. Sometimes an adventitious bursa may develop beneath it
3. If infected → an abscess is formed

-Treatment:

1. Avoid the causative agent if possible.
2. Shave the callosity with a razor blade and repeatedly painted with an ointment containing salicylic acid.



Corn

- Similar to a callosity but a combination of friction & pressure

-Example: tight shoes → down growth of a hard horny plug into the corium → pressure on sensory nerve endings → much pain.

- Treatment: as a callosity (Excision & suturing rarely necessary)



Wart

- Etiology: viral infection

- Mode of transmission:

Direct contact (gains access through abrasion)

- Pathology: localized overgrowth of epidermis & papillae of skin

- Clinically: a small horny projection and may be multiple

-Treatment: either 1. Curettage & diathermy

2. Repeated application of glacial acetic acid.

3. Cryosurgery using either liquid nitrogen or nitrous oxide



Keratoacanthoma (molluscum sebaceum)

- Etiology: exposure to the sun.

-Type of patient: middle aged persons

- Pathology: composed of keratinizing squamous cells.

- Clinically a firm, rounded reddish papule, enlarges rapidly over

8 weeks → a rounded, slightly umbilicated nodule **1-2cm** in diameter

-The centre contains a horny plug covered by a crust concealing a keratin filled crater

-Course:

Spontaneous healing takes **6 months** by shedding of horny core.

-Clinical importance:

Mimic an epithelioma or a rodent ulcer → unnecessary surgery

-Pathological examination:

Abrupt transition from normal to hyperplastic epithelium



Vascular anomalies

Haemangioma (Previously name→ strawberry mark)

Definition

Benign proliferative neoplasm of vascular spaces endothelial lining

Incidence

- Affects **10%** of white infants
- Female to male ratio is **3:1**
- The majority of lesions affect the head and neck area

Stages

1. Stage of proliferation (shortly after birth)

- A reddish patch that increases in size over the few following months

2. Involution phase (starts at the end of the 1st year)

- C/P: Central pallor appears & color of lesion slowly fades
- There is progressive decrease in the size of the lesion.
- Continuous until the age of **10 years**.

3. Involved phase

- The skin becomes normal in about **50%** of patients.
- Some residual changes as discolouration, scarring or telangiectasia may persist.

Complications

- 1- A periorbital haemangioma → may encroach on visual field
- 2- Serious bleeding from a ruptured large liver haemangioma.
- 3- Large cutaneous or visceral haemangiomata → a) congestive HF
b) Entrap platelets → thrombocytopenia.

Treatment

- 1- Reassurance of the parents and observation are recommended (skin appearance after involution is better than the scar following excision)
- 2- When it is necessary to interfere the following are helpful:
 - a) Oral or intralesional corticosteroids.
 - b) Laser photocoagulation
 - c) Surgical excision

Multiple terms were used to describe vascular anomalies as "cavernous" and "strawberry".
- Current nomenclature is biologically based:

1.

Hemangioma

2. Vascular malformations

a) Low flow lesions

- i. Capillary malformation
- ii. Venous malformation

b) High flow lesions

- i. Arterial
- ii. Arterio-venous

c) Lymphatic malformations



Vascular malformations

- Definition: structural abnormalities in the blood vessels due to embryological error.

- Time of presentation: may be present at birth, but often later.

Investigations

1- Doppler ultrasound → differentiate high-flow from low-flow lesions

2- MRI and/or (MRA) (the most useful)

Assess extent of lesion & degree of involvement with surrounding

3- Angiography

Types

1. Capillary malformation (port-wine stain)

- Presentation: since birth and do not undergo involution.

- C/P: 1. Lesion is dark purple and is not raised above the surface

2. Applying pressure → blanching but color returns immediately after release.

3. Sometimes, takes the distribution of one of branches of trigeminal nerve, but does not cross the middle line.

4. Sometimes associated with similar lesions in the meninges

(**Sturge-Weber syndrome**)

- Treatment:

i) **Pulsed dye laser** (of choice during childhood)

Requires general anesthesia & multiple sessions may be needed.

ii) **YAG laser** during adulthood (Results not as good as in children)

2. Venous malformation

- Similar to haemangioma in clinical picture but usually appears later

- As it is a malformation → does not undergo involution

- Treatment:

i) **Percutaneous sclerosis** with hypertonic saline, 100% alcohol or sodium tetradecyl sulfate

(There is a high recurrence rate → multiple sessions required)

ii) **Surgical excision**

Preoperative embolization or sclerosis → facilitates hemostasis

iii) **Interstitial laser coagulation.**

3. Arterial malformations (cirroid aneurysm)

- Definition: abnormal development of arterial structures, including stenosis or hypoplasia, duplication and/or tortuosity.

- Site: in the scalp (mostly) especially in the temporal or occipital regions and may involve the underlying bone.

- C/P: 1. soft, compressible & pulsating swelling with a marked bruit.

2. Ulceration of underlying skin may occur → serious haemorrhage

- Treatment: - difficult as the swelling is supplied by multiple feeding arteries which have to be ligated

- Preliminary embolization of the feeding vessels may be tried

They grow proportionately with the child & do not regress or involute



4. Arterial venous malformations (AVMs)

- Definition: abnormal connections between arteries and veins without an intervening capillary bed (high flow lesion)
- Presentation: at birth but may not be evident until late childhood
- C/P: 1. Localized AVM → localized swelling with increased surface temperature (There may be palpable thrill or murmur)
- 2. Diffuse AVMs → growth disturbance or skeletal distortion.
- Treatment:
 - Excision one day after embolization of feeding vessels.
 - Extensive coverage by a free-flap may be needed.

Hereditary haemorrhagic telangiectasia is an AD disease characterized by multiple cutaneous, visceral and mucosal AVMs

5. Lymphatic malformations (LMs)

- Classification: microcystic or macrocystic
- May have a component of venous malformation.
- Site: The head & neck region or the axillae (majority of cases)
- Neck masses may extend into the mediastinum or pectoral area. –
- Consequences: congenital macroglossia, macrotia & macrocheilia (lip enlargement) (skeletal involvement may cause distortion)
- Treatment:
 1. **Percutaneous aspiration followed by sclerosis** → short-term improvement & usually requires additional treatment.
 2. **Surgical excision** (multi-staged procedures)
- Technically easier as child grows (wait until at least **3 years** of age)



lipoma

Definition

A benign tumour that is composed of fatty tissue arranged in lobules.

Pathology

Gross picture

- The tumour has a lobulated surface and a yellowish color
- Enclosed in a false thin fibrous capsule → can be enucleated

Microscopic picture

- May contain excess fibrous tissue (**fibrolipoma**), angiomatous tissue (**angiolipoma**) or myxomatous tissue (**myxolipoma**)

Course → grows very slowly



Clinical picture

- 1- A solitary well defined swelling.
- 2- Multiple lipomatosis → multiple lipomas in limbs or trunk (differential diagnosis of multiple swellings)

Classification According to the site

1-Subcutaneous lipomas (the commonest)

- Characters: a) Slowly growing
- b) Painless and is not tender.
- c) Lobulated surface → may attach to the skin at multiple points
- d) Consistency is soft
- Some lipomas especially in warm weather may give pseudo fluctuation → due to: i- mobility of the tumour in its bed
ii) Fat at warm temperature may undergo liquefaction
- e) Well-defined & slippery edge as tumour moves inside the capsule
- f) The swelling is mobile over deep structures.

2- Subfascial lipomas (under the deep fascia)

- Not attached to skin & not have slippery edge → difficult diagnosis

3- Intermuscular lipomas

- Swelling is masked by overlying muscles → difficult to diagnose
- Fixed deeply
- When the muscle contracts → disappear (become more prominent if they are pushed out of the muscle)
- Differential diagnosis: soft tissue sarcoma → hard & grows rapidly

4- Submucous lipomas

- Site: larynx, stomach or intestine.
- A submucous lipoma in the larynx → respiratory obstruction.
- A submucous lipoma in the intestine → intussusception → I.O.

5- Parosteal lipomas

- Arise in relation to the skull and may cause bone erosion.

6- Extradural lipomas found within the spinal canal → paraplegia

7- Intra-articular lipomas

Complications

- 1- Degeneration → calcification.
- 2- Malignant transformation (very rare) may occur in retroperitoneal

Treatment

- A subcutaneous lipoma is usually excised for cosmetic reasons.
- The operation: enucleation of the tumour from inside its capsule



A lipoma is generally a very innocent tumour which does not cause any problem except in:

- a) Submucous lipoma in the intestine
- b) Extradural lipoma of the spinal canal

Benign lesions of melanocytes (naevi)

- Origin: develop from the neural crest and migrate to lodge between the basal cells of the epidermis.
- Their number /unit area of skin is fixed irrespective of race or skin coloration (skin color differences are due to melanin granules amount)
- Melanoma: true malignant neoplasm that arises from melanocytes.
- Melanocytes can give rise to various hamartomas

Types

1- Lentigo

- Melanocytes replace basal layer of epidermis over a certain area.

- Histologically:

Rounded cells containing fine granular brown melanin pigment

-Clinically: a pigmented well-defined spot varying in color from pale brown to black.

2- Junctional pigmented nevus

More proliferation of melanocytes → small nodules of epidermis which bulge downwards into the dermis

-Clinically: looks like a lentigo

3- Compound pigmented nevus

- Eventually the basement membrane is disrupted → some melanocytes pass to the dermis → lose their capacity to form melanin and are called nevus cells.

-In the dermis, a varying number of macrophages containing melanin pigments are present.

- C/P:

1. Raised above surface & vary in color from pale brown to black
2. Sometimes dark hairs are seen growing from the lesion surface

- Rarely, an extensive area of the skin is replaced by such hairy pigmented nevus (giant hairy nevus)

4- Intradermal pigmented nevus

- Around the age of puberty, the majority of instances junctional activity ceases & nevus cells in the dermis undergo maturation.

- Most pigmented naevi of adults are of this type.

-Clinically: look like compound pigmented naevi



Naevi that may turn malignant

1. The giant hairy pigmented naevi.
2. After puberty any pigmented nevus with junctional activity has the potential to undergo malignant changes (but rare)

Indications for surgical excision

- 1- For cosmetic reasons.
- 2- If they are subjected to repeated trauma, e.g. during shaving.
- 3- If there is suspicion of malignant transformation

Adequate surgical excision should be performed and the specimen should be examined histologically.

Precancerous skin lesions

Squamous keratosis (actinic or senile keratosis)

- Site: sun-exposed parts particularly in fair-skinned people.
- C/P: dry, rough, inelastic and irregularly pigmented areas of skin.
- Histologically: areas of hyperkeratosis, nuclear pleomorphism and increased mitosis with irregular cellular differentiation.
- The most important precursor of invasive squamous cell carcinoma



Bowen's disease

- Site: in non-exposed skin.
- C/P: sharply demarcated, rounded, reddish patches slowly growing
- Histologically: epidermal hyperplasia with cellular differentiation
- This disease is usually treated for a long time as psoriasis.



Malignant neoplasms of the skin

Basal cell carcinoma (rodent ulcer)

Incidence

- The commonest malignant lesion of the skin
- More common in males above the age of 40 years
- Persons with a light colored complexion are more than those with dark skin as the presence of melanin pigment protects against effects of ultraviolet rays.

This is a locally malignant lesion which arises from the basal cells of the epidermis

Etiology

- 1- Prolonged exposure to sun ultraviolet rays (**the most important**)
→ more common in farmers, sailors and countrymen in sunny areas
- 2- Albinism and xeroderma pigmentosum predispose to multiple basal cell carcinomata all over the body.
- 3- Ionizing radiation
- 4- Patients receiving immunosuppression

Pathology

Site:

- In the face in **90%** especially above a line from the lobule ear to the angle of the mouth.
- The commonest: a) outer or inner eye canthus b) Nasolabial fold

Gross appearance

- Starts as a small nodule covered by thin epidermis → ulcerates → serous discharge and bleeding
- Occasionally there are multiple lesions



- Later the patient usually presents by either

i) an ulcerated mass

Edge: rolled in like a car tyre and is beaded

Rate of growth: very slow (after 1 year → diameter ↑ only 1cm)

Floor: red granular and often covered with a dry crust or scab.

ii) The excavating type

- Ulcer erodes deeply into underlying structures → destruction & infiltration of the nasal sinuses

iii) Other less common types

a) The cystic

b) The pigmented (containing melanocytes)

c) The flat superficial spreading (presents as a red scaly patch resembling psoriasis or eczema)

Microscopic appearance

- The tumour cells are arranged as sheets (**Pallisade appearance**)

a) The outer layer → low columnar cells with tall nuclei

b) Inside this layer → polyhedral cells with large basophilic nuclei

- No tendency to keratinization & mitotic figures are uncommon

Spread

Only Direct spread to the surrounding & underlying structures

- If the draining LNs are enlarged = 2^{ry} infection or presence of a focus of basosquamous carcinoma

Diagnosis

By a biopsy

- If the lesion is small → excision biopsy

- Large lesion → a small piece of edge with an area & adjacent skin

Treatment

1) Radiotherapy (BCC is very radiosensitive)

- The dose is fractionated over several weeks.

- Contraindications: a) involvement of underlying cartilage or bone
b) The lesion is near the eyes

2) Surgery, indications:

1- A small lesion as it is easy to close the defect.

2- Infiltration of underlying cartilage or bone as:

i) Will protect malignant cells from the effect of radiotherapy

ii) Radiation cause necrosis of involved bone & cartilage

3- Radio resistant lesions.

4- Recurrence after previous irradiation

- Safety margin: 0.5cm is adequate regarding surface & depth

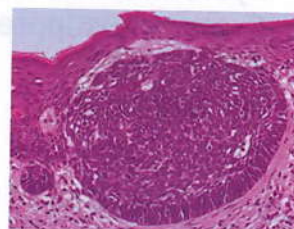
- Defect created after excision is closed by 1^{ry} suture or by flaps.

- The pathologist should check that an adequate safety free margin has been excised, and if not → re-operate or give radiotherapy

3) Other lines

a) Cryosurgery

b) Local application of cytotoxic drugs as 5FU cream (**Efudex**)



**Prognosis
Excellent**

- If the lesion has been completely excised → cure rate is **100%**.

- Recurrence is due to leaving behind foci of malignant tissue (more liable to occur if there is infiltration of bone or cartilage)

Squamous cell carcinoma (epithelioma)

Incidence More common in males especially elderly persons

Etiology

- 1- Prolonged exposure to sun ultraviolet rays (as in rodent ulcer).
- 2- Previous irradiation.
- 3- Albinism and xeroderma pigmentosum.
- 4- Long-standing irritation of the skin as in chronic granulomas, chronic ulcers, osteomyelitis, sinuses & old burn scars.
- 5- Prolonged exposure to carcinogenic agents as polycyclic hydrocarbons, coal tar derivatives or mineral oils.
- 6- Immunosuppression

Pathology

Site

- In upper part of the face, lower lip & dorsum of the hands
- Sometimes arises at the mucocutaneous junction

Gross appearance

- An ulcerated mass which grows rapidly
- Edges: raised and everted
- Floor: occupied by malignant fungating tissue.
- Base: indurated & it becomes rapidly fixed to underlying tissues.
- 2nd infection may occur & there may be blood-stained discharge

Microscopic appearance

A) Differentiated

- Characteristic appearance → '**epithelial pearls**' or '**cell nests**' (Outer prickly cells and keratin in the centre)
- As carcinoma cells enlarge, older cells in the centre produce some keratin → lose their organelles, become flattened & cornified → lie concentrically in the centre of the mass
- Infiltrate the underlying connective tissue

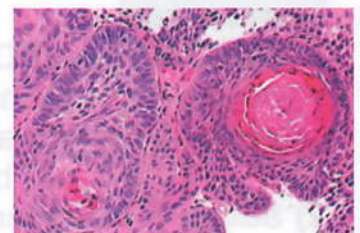
B) In undifferentiated tumours

- Cells are anaplastic with basophilic cytoplasm (no origin evidence)

Spread

- 1- Direct to adjacent structures (rapidly)
- 2- Lymphatic to regional LNs (Sometimes the nodes are enlarged due to 2nd infection)
- Fibrosis associated with scarring in Marjolin's ulcer makes lymphatic dissemination late
- 3- Blood stream spread is very uncommon

A carcinoma on top of a scar is called **Marjolin's ulcer** (usually there is delay in its diagnosis)



Differential diagnosis

1. Kerato-acanthoma
2. Basal cell carcinoma.
3. Malignant melanoma.

Treatment

A) Surgery

- Indications:

- 1- Small lesions → excision biopsy with an adequate safety margin.
- 2- Infiltration of cartilage or bone.
- 3- Radioresistant lesions.
- 4- Recurrence after previous radiotherapy
- 5- Marjolin's ulcer.
- 6- Block dissection of metastatic lymph nodes

B) Radiotherapy

-Indication: tumours of the head & neck particularly for poorly differentiated lesions (The resulting scar is fragile)

- The safety margin for epithelioma is at least 2 cm except in the face where it is 1 cm
- **Prognosis** → 90% five year cure rate

Melanoma

Incidence

- Increasing in Western countries (may be due to defective ozone)
- Almost unknown before puberty.

Etiology

- 1- Prolonged exposure to ultraviolet rays of the sun
- 2- Albinism and xeroderma pigmentosum.
- 3- On top of a benign nevus



Pathology and clinical picture

	Superficial spreading melanoma	Nodular melanoma	Lentigo maligna (Hutchinson's melanotic freckle)	Acral-lentiginous melanoma	Amelanotic melanoma
Incidence	64 %	12-25 %	1-15 %		
Site	In any part of the body	In any part of the body	In the face	Palms, soles & beneath the nail	
Age	Usually in middle age	Usually in younger age groups	Elderly persons		
Character of the lesion	Raised above the surface with irregular edge	- Raised above surface, grey or black in color & liable to ulceration	Begins as a flat brown macule which grows very slowly. -Some areas may even regress		
Prognosis	Good (as malignant melanocytes spread along the epidermis)	Bad as malignant cells invade the dermis	Least malignant (as malignant cells spread along basal layer of epidermis)	Poor	Very poor

Prognostic factors

- 1- Thickness of the tumour (Breslow classification)
- 2- Depth of invasion of the skin (Clark's level of invasion)
 - a) Epidermal → **the best prognosis**.
 - b) Dermo-epidermal junction.
 - c) Superficial papillary dermis.
 - d) Deep papillary dermis.
 - e) Subcutaneous tissue → **the worst prognosis**

Spread

- 1-Direct to SC tissues and the deep fascia
- 2- Lymphatic → by permeation or embolism
 - Lymphatic permeation → satellite nodules around the tumour or between the tumour and the regional LNs
- 3- Blood → to lungs, liver, bones, brain, & skin.

Diagnosis

By histological examination (The only sure method)

How to take the biopsy?

- 1- Line of incision should meet the possible subsequent excision.
- 2- Safety margin should be 3 mm.
- 3- Should include the whole skin & SC tissues to allow pathologist to determine Clark's level and Breslow thickness.
- 4- Paraffin section is better than frozen section.

Treatment

Surgical excision of the 1^{ry} lesion with an adequate safety margin of skin & SC tissue, but not including the deep fascia

- If the tumour thickness is:

- a) <1 mm → safety margin is 1 cm.
- b) 1-4 mm → safety margin is 2 cm.
- c) >4mm → safety margin is 3cm.

- If the regional LNs are:

- a) Enlarged & firm → radical block dissection
 - b) Free by clinical examination → sentinel LN biopsy
- Prophylactic block dissection is no longer performed.
 - Metastases treated by chemotherapy, interferons & interleukin-2.

Malignant skin tumours of vascular origin

Kaposi's sarcoma

- Origin: from the endothelium of lymphatic & blood vessels.

- May occur as:

- a) Sporadic
 - b) Endemic (in certain countries in Africa and may have a relation to cytomegalic virus (CMV) infection)
- One of immunosuppression manifestations associated with AIDS

- The thicker & deeper the lesion → the worse are the spread and prognosis

- Malignant melanoma may rarely arise in eye, meninges, or at the mucocutaneous junction as the anal canal

- Criteria of malignant transformation of benign nevus:

- 1- Increase in size or thickness.
- 2- Change of pigmentation.
- 3- Occurrence of itching, tingling, ulceration or bleeding.
- 4- Development of satellite nodules

- Differential diagnosis

1. Granuloma
 2. Pigmented BCC
 3. Haemangioma.
 4. Compound or junctional naevus
- Malignant melanoma can spread virtually to any organ (2^{ry} deposits are usually black)

Muscles tendons and fascia

Carpal tunnel syndrome

Incidence

The commonest type of nerve compression syndromes

Etiology

- Conditions that increase tension under the retinaculum as:
 1. Pregnancy
 2. Myxedema
 3. Acromegaly
- 4. Rheumatoid arthritis involving synovial sheaths of flexor tendons
- May be idiopathic

Clinical picture

Type of patient → mainly middle aged females

Symptoms

Early:

Pain in the thumb & lateral 3 fingers (gets worse by night)

- Increases by: fully flexing the wrist.
- Relieves by: injecting the tunnel with lidocaine & corticosteroids and splinting the wrist at night
- The little finger is never affected in this syndrome.
- Palmar branch of median nerve escapes compression (passes superficial to retinaculum) → palm is not affected

Late

- 1- Paraesthesia & altered sensation of skin supplied by median N.
- 2- Weakness & atrophy of thenar eminence muscles.

Signs

1- Special tests:

a) **Tinel's percussion test** → Percussion over the carpal tunnel → tingling along the distribution of the median nerve.

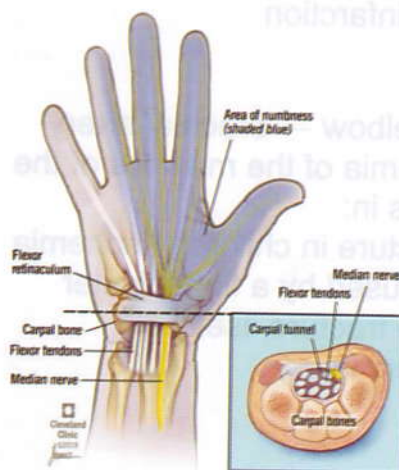
b) **Phalen's test**

- Flexion of wrist for one minute → pain along median n. distribution

2- Nerve conduction study → delay of median nerve conduction

Treatment

- In early cases → steroid injection → relieves symptoms
- Surgical division of the flexor retinaculum → pleasant results (The operation can be performed by endoscopy)



Anatomy

Median nerve is compressed as it passes through the carpal tunnel, in front of wrist.

- Boundaries of the tunnel:

a) Posteriorly:

carpal bones

b) Anteriorly:

flexor

retinaculum

- Contents:

a) Median N.

b) 8 tendons of flexor digitorum superficialis & profundus (within ulnar bursa)

c) Flexor pollicis longus tendon (within radial bursa)

- After passing deep to the retinaculum into hand, gives:

a) Motor supply → 3 muscles of thenar

eminence & lateral two lumbricals

B) Sensory supply → lateral 3 & 1/2 fingers.

- Palmar branch of median nerve arises above wrist superficial to flexor retinaculum

Dupuytren's contracture

Also known as "palmar fasciitis"

Incidence

Males are affected **10 times** more than females

Etiology

- 1- Idiopathic
- 2- Some cases are familial
- 3- More in cirrhotics, alcoholics, epileptics under phenytoin treatment & diabetic

Clinical picture

- Affects primarily the medial side of the palmar aponeurosis.
- The disease is bilateral in **1/2** of the cases.
- Starts as a nodule at base of ring or little finger followed by thickening and contracture → throws the finger or fingers into flexion:
 - a) The skin gets adherent to the fascia and contracts as well.
 - b) The other fingers progressively become involved.
 - c) Late cases involve the capsules of the metacarpo-phalangeal & PIP joints → their flexion
 - d) The DIP joints are free
- Palpation of the palm → a firm, irregular shaped nodule, **1-2 cm** proximal to the base of ring finger

Treatment

- Early cases → physiotherapy
- Late cases → Surgery, varies from:
 - a) Minor subcutaneous fasciotomy
 - b) Extensive operation → excision of the aponeurosis, joint capsulotomy and skin grafting to cover the resulting defect



The flexion deformity is not lessened by flexing the wrist joint (Opposite to Volkmann's ischaemic contracture)

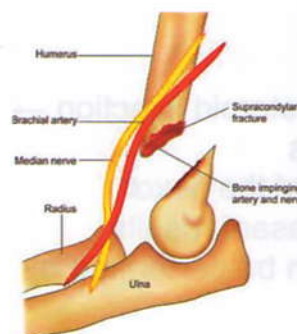
Volkmann's ischaemic contracture

Definition

Fibrosis and shortening of muscles of the front of the forearm due to acute ischaemia and infarction

Etiology

- Injuries around the elbow → brachial artery injury → acute ischaemia of the muscles of the front of the forearm as in:
 - a) Supracondylar fracture in children (ischemia is more commonly caused by a tight plaster rather than due to the fracture itself)
 - b) Direct brachial artery injury
 - c) Arterial embolism



- Acute muscle ischaemia → infarction after **6-12 hours**
- Muscle fibrosis & shortening develop within a few months

Clinical picture

A) Early (while the arm is immobilized for fracture treatment)

- 1- Pain in the forearm muscles and hand
- 2- Passive extension of the fingers is painful and limited.
- 3- Fingers & hand are pale, cold & show sluggish capillary circulation
- 4- The radial pulse may be absent.
- 5- If the median nerve is ischaemic → paraesthesia or severe burning in its distribution.

B) Established contracture

- 1- Pain is lessened but typical deformity appears
- 2- The forearm and hand appear wasted.
- 3- All the finger joints are flexed (**claw hand**).
- 4- Extension of the fingers is limited but improves as the wrist is flexed (compare Dupuytren's contracture).



Differential diagnosis of claw hand

- 1- Volkmann's ischaemic contracture.
- 2- Combined median and ulnar nerve injuries.
- 3- Lesions affecting lower roots or medial cord of brachial plexus (As Klumpke's paralysis & malignant infiltration of brachial plexus)
- 4- Advanced rheumatoid arthritis.
- 5- Spinal cord lesions as syringomyelia and poliomyelitis

Treatment

A) Early cases → Physiotherapy by gradual stretching & splinting

B) Late cases → The following procedures may be tried:

- 1- Muscle slide operation → the common flexor origin is detached from the medial epicondyle and is fixed at a lower level.
- 2- Tendon lengthening and tendon transfer.

Chronic tendonitis

Tennis elbow

Symptoms

Pain in the elbow at rest and worsens when he uses his hand.

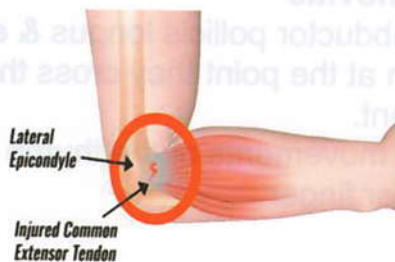
Signs

The attachment of extensor muscles of the forearm to the lateral epicondyle is tender

Etiology → direct trauma or repeated athletic activity

Treatment

- 1- By rest
- 2- In resistant cases → local injection of corticosteroid and local anaesthetics



- Appearance of early features in a patient with a fracture or after plaster cast application is very serious because at this stage the ischaemia can be corrected and the muscles saved

- Hand grip is weak (all the muscles have some function, whereas when a claw hand is due to nerve lesion → some muscles will be completely paralysed)

- Prevention

- Prompt restoration of muscle blood supply before development of infarction by:

- a) Reduction of fractures and dislocations
- b) Release of tight plaster cast
- c) Removal of constrictions

- If the radial pulse does not return → explore the brachial artery & the injured segment is excised & reconstructed by a vein graft

Rotator cuff (supraspinatus) tendonitis

-Incidence:

- 1- The commonest cause of shoulder pain with movement limitation
- 2- Any of rotator cuff muscles may be involved (the commonest is the **supraspinatus** tendon)

- **Etiology**: repeated trauma from sports or occupational activities

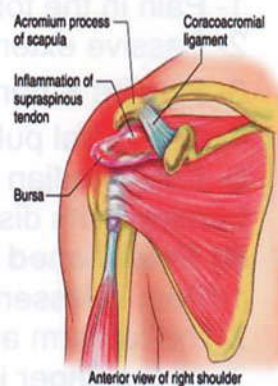
- Inflammation often extends to affect the subacromial bursa.

- **Symptoms**: painful active abduction (particularly when the shoulder moves between 60° & 120°) → due to compression of inflamed rotator cuff & bursa beneath acromion → known as "**painful arc syndrome**".

-Treatment:

A) Conservative using NSAID & immobilization followed by gradual active exercise to restore the full range of shoulder movement

B) Resistant cases → local injection of corticosteroid lidocaine mixture



Stenosing tenovaginitis

- **Cause**: fibrous stricture in a tendon sheath.

- **Examples**:

I-Trigger finger

- **Pathogenesis**: thickening of fibrous flexor sheath at level of the metacarpo-phalangeal joint → pain on tendon movement & local tenderness.

- **Clinical picture**:

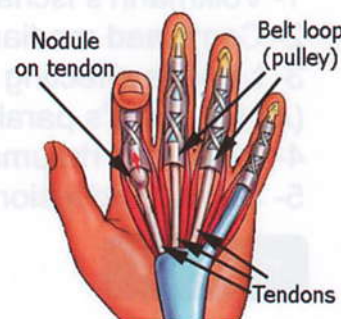
Symptoms: When the finger is flexed → swelling of the tendon proximal to the constriction → interferes with finger

- Attempts to extend the finger may require passive assistance → tendon snaps through the narrow area → likened to the snap of the trigger of a pistol → therefore called "**trigger finger**".

Signs: thickening of tendon or tendon sheath at the level of the head of the metacarpal bone.

- **Treatment**: 1-Early cases → splinting and steroid injection.

2- Late cases → division of the constricting fibrous flexor sheath

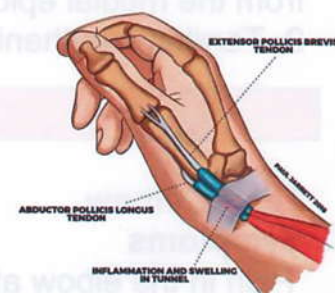


II- DeQuervain's synovitis

- Inflammation of the abductor pollicis longus & extensor pollicis brevis tendon sheath at the point they cross the wrist → pain and limitation of movement.

- Active and passive movements of the thumb exaggerate the pain.

- **Treatment**: as trigger finger



Chronic bursitis

Definition of bursa

Fluid-filled cavities lined with flattened epithelium similar to synovium

Types

1- Anatomical

2- Adventitial

Anatomical bursae

- Function: to allow easy movement between tendons, bones & skin

- Etiology of chronic inflammation: f repeated friction

- Clinical picture: 1- mildly painful cystic swellings

2- Crepitus may be noticed (if the bursa lining is rough or the fluid contains small loose fibrinous particles)

- Common sites:

1- **Prepatellar bursitis** (**House-maid's knee**)

- Site: over the lower half of the patella behind the skin.

2- **Infrapatellar bursitis** (**Clergyman's knee**)

- Site: between ligamentum patella and the head of the tibia

- When ligamentum patella is distended → a fluctuating swelling appears on either side of the ligamentum.

3- **Olecranon bursitis** (**student's elbow**)

- Site: between the skin and olecranon

4- **Semimembranosus bursa**

- Site: between the semimembranosus tendon and the posterior aspect of the medial femoral condyle.

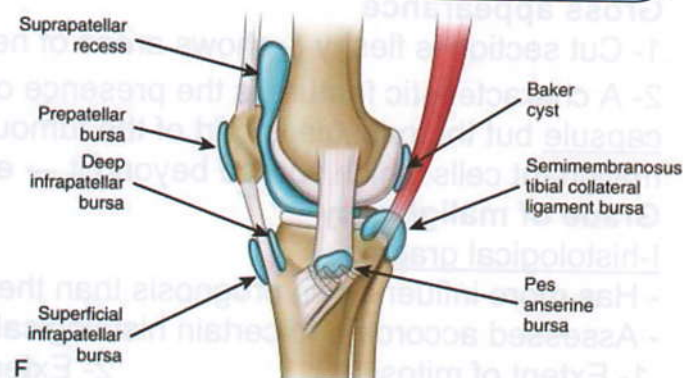
- Type of patient: usually a middle-aged adult

- Symptoms: a swelling behind knee joint

- Examination: a) cystic swelling on medial side of the popliteal fossa just above the joint

b) Contraction of semimembranosus muscle during knee flexion → the swelling diminish in size

- Differential diagnosis: Baker's cyst



Baker's cyst

- Definition: a pulsion diverticulum of the knee joint

- Type of patients: 1. with rheumatoid arthritis or osteoarthritis.

2. More in elderly patients

- C/P:

1. presents as a cystic swelling below the level of the knee joint and deep to Gastrocnemius muscle

2. Reduce into the joint (characteristic finding)

Adventitious bursae

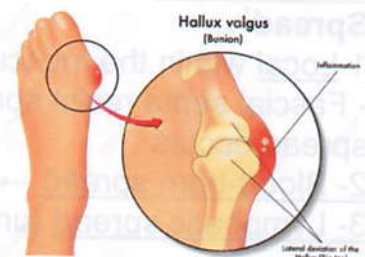
- Site: in any site of friction between two layers of tissue.

- Example: **the bunion** → develops between the skin & head of the 1st metatarsal (particularly with the hallux valgus deformity) •

- Treatment:

1- If friction can be abolished → bursitis may regress

2- If can't → excision & correction of any underlying deformity



Soft tissue sarcomas

Definition

Malignant tumours arising in the extraskeletal connective tissue

Incidence

1 % of all malignant tumours (uncommon)

Etiology

Exact etiology unknown but some disorders are associated with certain types of soft tissue sarcomas as:

- 1- Lymphangiosarcoma: in the chronic post mastectomy arm edema
- 2- Neurofibrosarcoma: with von Recklinghausen's disease

Pathology

Site

- 1- Extremities & pelvic girdle (in majority)
- 2- Trunk and retroperitoneal tissues

Types and histological picture

- Origin: primitive multipotential mesenchymal (connective tissue) cells which differentiate during neoplastic transformation.

- | | |
|-----------------------------------|---------------------|
| 1- Malignant fibrous histiocytoma | 2- Fibrosarcoma |
| 3- Malignant nerve sheath tumour | 4- Leiomyosarcoma |
| 5- Liposarcoma | 6- Rhabdomyosarcoma |
| 7- Angiosarcoma | 8- Synovial sarcoma |

Gross appearance

- 1- Cut section is fleshy & shows areas of necrosis & haemorrhage
- 2- A characteristic feature is the presence of a well defined false capsule but the capsule is part of the tumour & infiltrated with malignant cells which spread beyond it → enucleation is obsolete

Grade of malignancy

I-histological grading

- Has more influence on prognosis than the type of the tumour.
- Assessed according to certain histological criteria which include:
 - 1- Extent of mitosis
 - 2- Extent of necrosis
 - 3- Cellular anaplasia
 - 4- Pleomorphism
- The tumour is then judged to be low, intermediate or high grade

II- DNA ploidy

Spread

- 1- Local within the musculofascial compartment in which it arises
- Fascial septa resist spread temporarily then extra-compartmental spread occurs
- 2- Blood-born spread → mainly to the lungs
- 3- Lymphatic spread (unusual)

DNA ploidy

- Used to grade these tumours.
- A normal cell is **euploid** (contains 46 chromosomes "2n")
- Malignant cells are usually **polyploid** (contain 4n, 8n, etc.) and **aneuploid** (bizarre chromosomal content).
- Deviation of a tumour from normal ploidy can be assessed Using a "**flow cytometer**" → determine the tumour aggressiveness

Clinical picture

- Type of patient:

- 1- In all age groups including children
- 2- The commonest in 5th & 6th decades of life

- **Symptoms:** a painless swelling which has been gradually enlarging over several months without disability → therefore delay presentation

- **Signs:** soft or firm in consistency (depending on the amount of deposited collagen)



Investigations

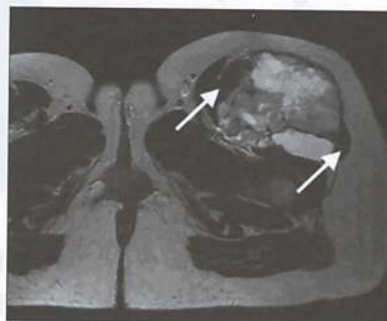
A) Imaging

- 1- CT scan or MRI (of choice)

Show the tumour extent & its relationship to muscle groups

- 2- Chest X-ray and CT scan

Detect pulmonary metastases



B) Tissue diagnosis

- **Aim:** obtain histological confirmation of the diagnosis before planning treatment

- **Methods:**

- 1- **Ture cut needle biopsy** (under local anaesthesia)

- 2- **Open biopsy** → when cannot reach diagnosis after needle biopsy

Treatment

A) Operable cases

- The standard is combination of:

- 1- Radical surgical excision with adequate safety margin

(If radical resection will leave a useless limb → amputation)

- 2- Adjuvant radiotherapy

B) Inoperable cases

- **As patients with:**

- 1- Pulmonary metastases

- 2- Huge retroperitoneal sarcomas that are adherent to important irremovable structures.

Considered to be beyond cure

Differential diagnosis

- 1- Benign soft tissue tumours, e.g., a lipoma.
- 2- Deep seated haematoma
- 3- Malignant LNs
- 4- Bone tumour
- 5- Tumour like conditions of soft tissues:

A)

Fibromatoses

- **Best example:**

desmoid tumour (Paget's recurrent desmoid tumour)

- **Site:** in the rectus sheath & the external oblique aponeurosis

- **Type of patient:** middle aged women.

- **Growth rate:** very slow

- **Treatment:** wide surgical excision as a low grade soft tissue sarcoma as it has tendency for recurrence after an apparently adequate excision

B) Nodular & proliferative fasciitis

C)

Proliferative myositis & myositis ossificans

The hand and foot

Tendons

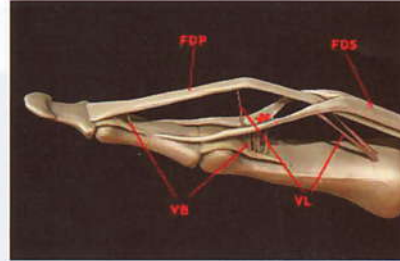
- Cross the wrist either: a) ventrally (flexors) b) dorsally (extensors)
- Function: to move the wrist & fingers
- The long flexor tendons arise from two muscles:

a) The flexor digitorum superficialis (sublimis)

- Each superficialis tendon splits opposite middle phalanx forming a tunnel & inserts at the sides of the anterior surface of this phalanx

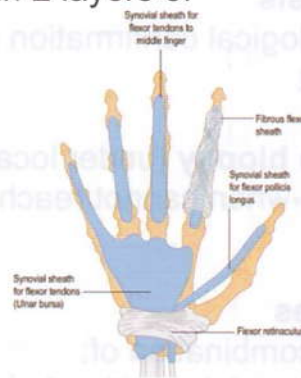
b) The flexor digitorum profundus

- The tendon passes through superficialis tunnel & crosses DIP joint to be inserted in the front of base of the distal phalanx (Superficial muscle does not flex DIP joint while the profundus can move all joints which it crosses)



Synovial sheaths

- Function: allow free frictionless movement of the hand
- The flexor and extensor tendons are enclosed in 2 layers of synovium separated by fluid
- Opposite each finger, the flexor tendons are enclosed in a digital synovial sheath that is covered by a fibrous flexor sheath.
- The radial bursa → encloses flexor pollicis longus
- The ulnar bursa → encloses the superficial & deep flexor tendons of other fingers (in palm & distal forearm)
- Tendon sheath infections spread due to:
 - a) The radial and ulnar bursae commonly communicate
 - b) Ulnar bursae communicates with little finger synovial sheath



Intrinsic muscles

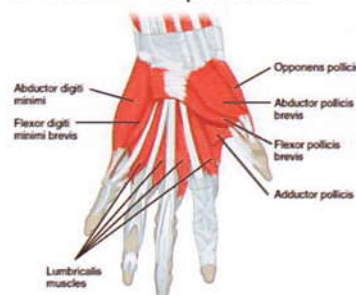
- 1- The thenar & hypothenar muscles → flexion, abduction & opposition of thumb and little finger respectively (The thumb has an additional adductor pollicis muscles)

- 2- The interossei:
 - a) Dorsal → abduct
 - b) Palmar → adduct

Metacarpo-phalangeal joint

3-The lumbricals

- Arise from → flexor digitorum profundus tendons
- Inserted with → long extensor tendons in the extensor expansion.
- The lumbricals and the interossei place the hand in the writing position (flexion of the metacarpo-phalangeal and extension of the interphalangeal joints)
- The thumb has no lumbricals



Bones

1- Carpus: is formed of 8 small bones arranged in two rows

2- Metacarpus: 5 metacarpal bones that articulate with the phalanges at metacarpo-phalangeal joints

3- Phalanges: The thumb is formed of 2 phalanges, while each of the other four fingers is formed of 3

-Flexor retinaculum

Extends transversely across the anterior concavity of the carpus converting it into a tunnel -Structures passing through this carpal tunnel to reach hand:
 a) The long flexor tendons with their synovial covering,
 b) Median nerve

Nerves

1- Median nerve

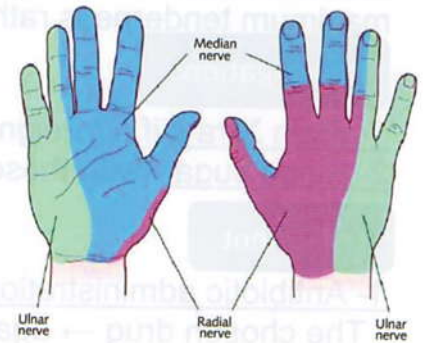
- Motor supply: a) thenar muscles (except the adductor pollicis)
b) The first lumbrical.
- Sensory supply: to the lateral 2/3 of the palm skin & the anterior surface of the lateral 3 ½ fingers

2- The ulnar nerve (the main motor supply of hand muscles)

- Motor supply: the rest of the intrinsic muscles
- Sensory supply: to front & back of medial 1/3 of the hand and the medial 1 ½ fingers

3- The radial nerve

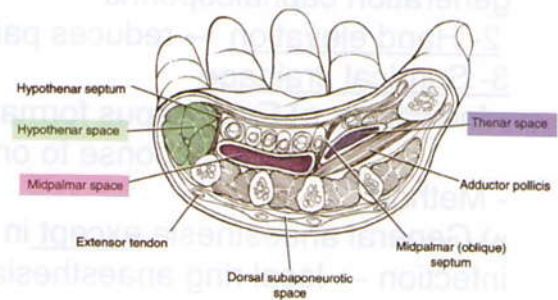
- Has no motor branches in the hand
- Sensory supply: the rest of skin of the dorsum of the hand fingers



Fascial spaces

The mid-palmar space, boundaries:

- 1- **Anteriorly**: the flexor tendons of the little, ring & middle fingers
- 2- **Posteriorly**: the fascia covering the interosseous muscles
- 3- **Medially**: fibrous septum separating it from hypothenar eminence
- 4- **Laterally**: a strong fibrous septum extends from the palmar fascia to the middle metacarpal bone (Separating it from the thenar space)



Arteries

- The radial and ulnar arteries

- Communicate through the superficial & deep palmar arches.
- Branches: digital branches that run on each side of the fingers, in a dorsal position to the corresponding digital nerves
- Because of the free communication between them → ligation or block of one of them does not affect the viability of the hand

Skin

a) The palmar skin: - Devoid of hair & sebaceous glands.

- Tough and adherent to underlying palmar aponeurosis.

B) The dorsal skin: - soft and rests on loose areolar tissue.

- Because of this difference, edema fluid collects on the dorsum.

Hand infections

Etiology

- Causative organism: Staphylococcus aureus (In 90% of cases)

- Route of infection: through minor injuries & punctures of the hand

Clinical picture

- Type of patient: prevalent in manual workers and house wives.
- Symptoms: pain, swelling & fever.
- Examination: site of infection is known by finding the point of maximum tenderness rather than the area of edema

Investigations

- 1- Plain X-ray: if a foreign body is suspected.
- 2- Blood sugar (with those recurrent infections) may reveal DM

Treatment

- 1- Antibiotic administration (immediately started)
 - The chosen drug → against Staph. Aureus as flucloxacillin, erythromycin, amoxycillin clavulanic acid combination & 1st or 2nd generation cephalosporins
- 2- Hand elevation → reduces pain and edema
- 3- Surgical drainage
 - Indication: a) Evident pus formation from the start
b) No response to one day intensive antibiotic therapy
 - Method:
 - a) General anaesthesia except in paronychia & distal pulp space infection → local ring anaesthesia (without adrenaline) at finger root
 - b) Apply a pneumatic tourniquet around the upper arm after hand elevation for a few minutes → obtain a clear bloodless field
 - c) Appropriate skin incisions and pus is drained by a sinus forceps.
 - d) Pus is sampled for culture and sensitivity.
 - e) Soft rubber drains are preferred, e.g. a piece of surgical glove.
- 4- Postoperative elevation, physiotherapy and wound dressings

Classification

A) Cutaneous and subcutaneous:

1. Paronychia
2. Subcuticullth & S.C. whitlow
3. Pulp space infection
4. Web space infection

b) Fascial spaces

1. Midpalmar space infection
2. Thenar space infection
3. Hypothenar space infection
4. Space of Parona infection

C) Synovial sheaths

1. Acute digital tenosynovitis
2. Ulnar bursitis
3. Radial bursitis

D) Bone and joint

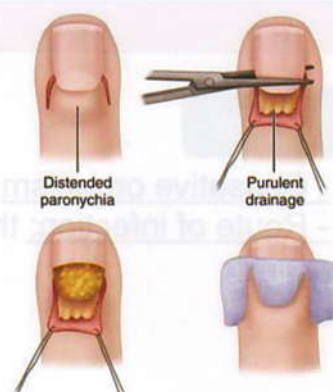
Paronychia

A) Acute paronychia

- Definition: Acute suppurative infection of the nail fold
- Incidence: the commonest form of hand infections.
- Treatment: Pus drainage using a fine tipped scalpel to raise the nail fold and to incise the skin cap through which pus points
 - If there is a subungual extension → excise the related part of nail

B) Chronic paronychia

- Type of patient: chronically wet nails as in dishwashers
- Associated fungal infection and nail deformities are common.
- Treatment:
 1. keeping the hands dry
 2. Antifungal therapy.
 3. Removal of the nail (in resistant cases)



Subcuticular & subcutaneous whitlow (abscess)

- Subcuticular whitlow: a subepithelial collection of pus
- Drainage by excision of the insensitive roof without anaesthesia
- A subcutaneous abscess requires anaesthesia for drainage
- Collar-stud abscess: abscess has two communicating components, subcutaneous and subcuticular

Pulp space infection (Felon)

- The commonly infected is → distal pulp space

- Anatomy

- Site: anterior to the distal phalanx
- Content: fatty tissue that is divided into locules by multiple septa running from skin to front of the bone
- Proximal boundary: insertion of profundus tendon to distal phalanx

- The digital artery divides in the middle segment of the finger into:

- a) Epiphyseal branch → enters the articular end outside pulp
- b) Diaphyseal branch → traverses the pulp.

- Clinical features and complications

- Edema → high rise of tissue pressure (as it is a tight compartment) → severe pain, if neglected → septic thrombophlebitis of the digital vessels → osteomyelitis of the shaft of terminal phalanx.
- Fortunately, regeneration can be obtained from undamaged proximal part which is supplied by the epiphyseal branch.

- Treatment

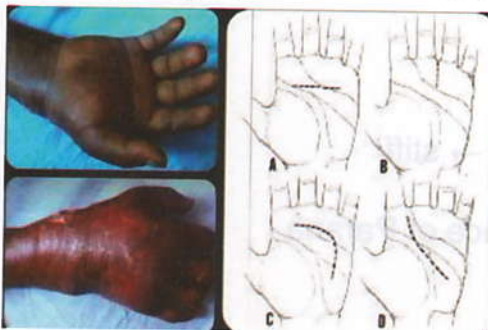
- Early surgical drainage (incision is over the most tender point)

Midpalmar space infection

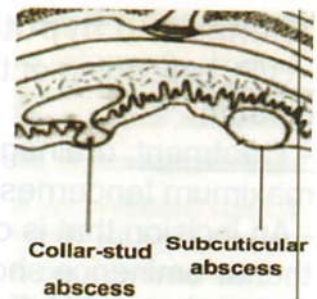
- Site: deep to the tough palmar aponeurosis
- Etiology: a) punctures
b) spread of web space or tendon sheath infection
- C/P: marked dorsal edema.

- Treatment: Pus evacuation through an incision in one of the transverse palmar creases.

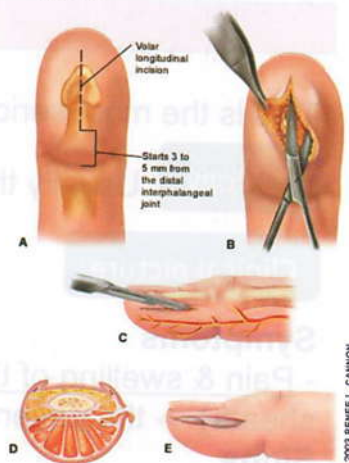
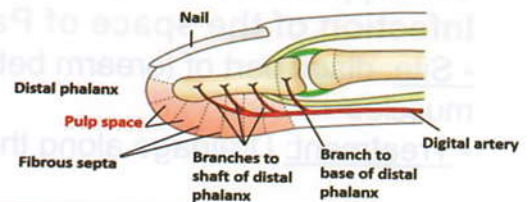
- To avoid injury of deep important structures, the incision is made through the skin only and then a sinus forceps is pushed inwards and is gently opened to let out pus (**Hilton's method**).



Web space abscess



Digital Pulp Space



© 2003 RENEE L. CANNON

"Don't wait for fluctuation in pulp space infection"

Web Space Infection

- C/P: swelling → separation of the 2 adjacent Fingers

- Complications:

Spread along the lumbricals to the mid palmar space

- Treatment:

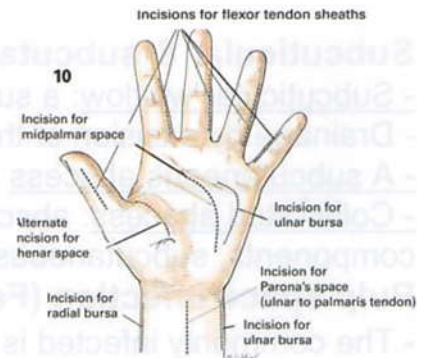
Pus evacuation through dorsal longitudinal incision between the fingers

Thenar and hypothenar spaces infection

- C/P: ballooning of thenar eminence & edema of hand dorsum

- Treatment: drainage through an incision at the site of maximum tenderness or where pus points at the skin.

- An incision that is done along the medial side of the thenar eminence should stop **2 cm** distal to the distal wrist crease (to avoid injury of motor branch of median nerve that supplies thenar muscles)



Infection of the space of Parona

- Site: distal part of forearm between pronator quadratus & flexor muscles

- Treatment: Drainage along the ulnar side of the forearm

Acute tenosynovitis

This is the most serious of hand infections

Etiology Usually the sequel of deep pin pricks

Clinical picture

Symptoms

- Pain & swelling of the finger (due to involvement of a digital flexor sheath) → the patient keeps it in the semiflexed position.

Signs

1- Active or passive movement → stretches the inflamed synovium → severe pain

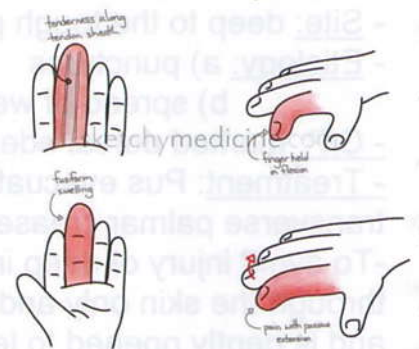
2-Tenderness is maximum just proximal to the crease over the metacarpophalangeal joints, which is synovial sac proximal extent

3-Little finger sheath affection → spread to ulnar bursa → marked hand swelling, semiflexion & limitation of movement of medial 4 fingers & diffuse palm tenderness (may extend to distal part of forearm → maximum point of tenderness may be on the ulnar side of the palm between the two palmar creases (**Kanavel's sign**)).

(Radial bursitis produces a similar picture in the thumb)



Suppurative Flexor Tenosynovitis (Kanavel's Signs)



Complications

1-Thrombo-phlebitis of the tendon vessels → sloughing → stiff useless finger

2- Infection spread to the mid palmar space and the space of Parana

Treatment

1- Conservative treatment

2- Surgical drainage

- Indication: Lack of response to intensive conservative treatment
- Incision: inflamed synovial sheath is drained through **2 incisions** → one at its proximal and another at its distal end.
- A fine catheter is inserted through each incision → allow drainage
- Irrigation of the sheath with an antibiotic solution

Ingrown toe nail

Definition

A sharp edge of the big toe nail impinges on the adjacent skin fold

Incidence

A common problem especially in young males

Predisposing factor

- 1- Faulty nail trimming (Oblique nail sides) → a sharp spike
- 2- Wearing tight → pushes this spike in the soft tissues

Complications

Infection (may be made worse by attempts of patient to cut the nail)

Treatment

A) Conservative treatment

- Indication: early cases
- The aim: lift off the nail spike
- Method:

- 1- A band of gauze soaked in an antiseptic is inserted beneath the ingrowing part of the nail → raise it up & alleviate the injured tissues
- 2- Thinning of nail centre makes it flexible & facilitates edge elevation
- 3- The bandage is changed daily until the nail grows past nail fold
- 4- The patient is instructed to avoid tight shoes & shown the proper method of square nail trimming (the centre of the nail is kept at the same level of its edges)

B) Surgery

- Indication: failure of conservative treatment & for suppurative cases
- Method: 1. Done under general or local anaesthesia.
- 2. A tourniquet is desirable to provide a clear bloodless field.
- 3. The side of the nail that is embedded is excised together with the infected skin & granulation tissue
- 4. The tough white germinal matrix is excised back to its origin (just distal to the interphalangeal joint)
- 5. Phenol application to ensure complete ablation of germinal matrix.
- 6. If the area is heavily infected, the wound is left open to granulate; otherwise its edges are loosely approximated



IN severe suppurative infection, Simple nail avulsion → rapid relief but recurrence is common with nail regrowth → **Wedge (segmental)** excision of germinal matrix is the definitive treatment

- The nail grows from germinal matrix that shows as a white crescent (**lunula**) under its proximal part → removal of lateral or medial part of germinal matrix prevents regrowth of the corresponding part of the nail

Hand injuries

Classification

1-Tidy (clean-cut) injuries

- Cause: by sharp objects as knives and broken glass.
- Skin is cleanly cut and tendons, nerves & vessels are affected

2- Untidy (crush) injuries

- Cause: by crushing forces and by burns
- Tissue devitalization (all hand tissues affected including bones)



Emergency

- Treatment of the multiple injury patients (ABCD)
- An amputated hand or digit is kept in a clean polythene bag, which is placed in a container full of cold water at 4°C (it should not be frozen) then urgently refer the patient to a centre that is equipped with microsurgery facilities.
- Prophylactic antibiotics & tetanus prophylaxis are administered

Assessment of injury

- Clinical & radiological assessment should include the skin, nerves, arteries, tendons and skeleton
- Inspect the wound & examine the hand for deformities, sensory and motor deficits.
- In many cases pain limits the value of these tests & final diagnosis of the extent of injury is obtained during surgical exploration

Treatment of minor injuries

1- Subungual haematoma

- Definition: a painful condition due to blood accumulation beneath the nail

- Treatment: - evacuating the accumulating blood by drilling a hole in the nail using the needle of a syringe.

- The procedure is painless → requires no anaesthesia.

2- A minor skin wound → stitched under local anaesthesia.

Principles of surgery for hand injuries

1- Anaesthesia

- a) General anaesthesia (preferred)
- b) Local intravenous anaesthesia with a tourniquet
- c) Nerve blocks with local anaesthetics

- Indication: for injuries of the distal parts of the fingers

- The brachial plexus, median or ulnar nerve may be blocked according to the site of injury

The digital arteries are end arteries and if they are thrown into prolonged spasm by the addition of adrenaline to the digital block → gangrene of finger

2- A tourniquet → clear bloodless field.

-It should be released within one hour

3- The use of magnifying loops or microscope is desirable.

4- Careful wound toilet: using saline irrigation, removes FB and debris.

5- Excision of devitalized tissues in untidy injuries.

6- Hemostasis by ligating small bleeding vessels

- An injured radial or ulnar artery can be ligated without affecting hand viability.

-If facilities & experience are available, repair of an injured artery is a better alternative.

- Arterial repair is necessity if both main arteries are injured.

7-Nerves

- In tidy wounds → 1st repair of cut nerves

-For untidy injuries or contaminated wounds → postpone repair after wound healing and resolution of inflammation.

8-Tendons (as nerve repair)

- Suturing with nonabsorbable nonirritant material as polypropylene

-Success of tendon repair is endangered by the development of adhesions which limit its mobility.

-Tendon grafting is reserved for complicated cases with loss of substance and for recurrent cases.

9- Bones and joints: should be managed either operatively by fixation (internal or external) or non-operatively by a cast or splint.

- Joint dislocations should be reduced

10- Finger tip injuries with skin loss

- In skin loss only → cover finger tip by split or full thickness graft.

- With finger tip amputations that expose the bone → Skin grafts will not take & local skin flaps are used to cover the defect (as cross finger flap or advancement of 2 lateral triangles of skin & S.C. tissue)

11- Skin closure (done without tension)

- Contaminated wounds are left opened (wounds of human and animal bites are highly contaminated with a mixture of bacteria including anaerobes & should never be sutured)

12- Microsurgical reimplantation of severed parts gives good results when it is done early for a clean-cut amputation (Useless if the part is severely crushed)

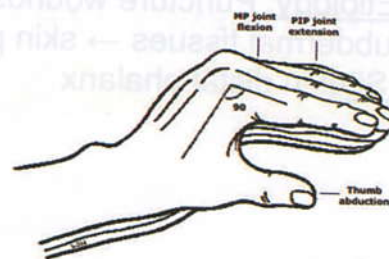
Postoperative care

1- The hand is bandaged in the position of function (the metacarpo-phalangeal joints are flexed while the interphalangeal joints are extended and the wrist is slightly extended)

2- Hand is kept elevated for few days → minimize edema formation.

3- Physiotherapy (started as early as possible) → allow restoration of hand function

- Tendon adhesion is most marked in flexor tendon injuries (in the area between the distal palmar crease and the middle of the fingers) where the flexor digitorum superficialis and profundus tendons are enclosed within the digital fibrous flexor sheath.
-With improvement of surgical technique, the tendency is now for direct suturing Irrespective of the site of injury



Swellings of the hand

Incidence

The great majority (94%) are benign

Simple ganglion

- **Definition:** a small cyst that contains a clear gelatinous fluid

- **Pathogenesis:**

- Protrusion of a joint or tendon synovial membrane → becomes isolated → form a cyst full of synovial fluid → fluid thickens to form the characteristic jellylike ganglion content.

- Synovial membrane protrusion may be an acute or a chronic

- **Site:** most commonly found on the dorsum of the wrist.

- **Clinical features**

- **Symptoms:** A painless small, rounded cystic swelling usually on the dorsum of the wrist (less commonly on the dorsum of the foot)

- **Signs:** tense cystic giving a false impression of being hard.

- A characteristic sign is that the mobility of the swelling is markedly restricted by stretching or by contraction of the related tendons

- **Treatment** (Not indicated unless the patient insists)

1- Aspiration or rupturing the cyst (by applying direct pressure) → temporary disappearance followed by recurrence

2- Excision (the only definitive treatment)

- Done under general anaesthesia & a tourniquet is applied

- Excised the wall should completely, otherwise recurrence occurs



Glomus tumour

- **Incidence:** a rare tumour

- **Origin:** heat regulating arteriovenous shunts & the unmyelinated nerves that control them.

- **Site:** anywhere in the skin but commonly found beneath a nail.

- **C/P:** markedly painful & tender and has a red or bluish color

- **DD:** 1. subungual haematoma 2. exostosis

3. melanoma

- **Treatment:** total excision under magnification



Implantation dermoid cyst

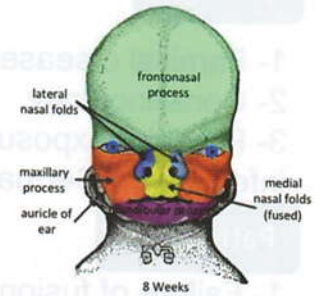
- **Etiology:** Puncture wounds → drive in small pieces of skin in the subdermal tissues → skin produces sebaceous material → a cyst.

- **Site:** in distal phalanx

Face, lips and palate

Development of the face

- The human face is formed by fusion of **5** embryonic prominences
- Each process is formed of a core of mesenchymal tissue lined by epithelium of endodermal origin and covered by surface ectoderm
- They encircle the stomodaeum (**primitive mouth**).
 - a) One fronto-nasal process (indented by two olfactory pits dividing it into a median and two lateral processes)
 - b) Two maxillary processes
 - c) Two mandibular processes (will form the floor of mouth, lower jaw and lower lip)



Development of the lips

- **Upper lip** → formed of the maxillary processes
- The middle part of upper lip (**philtrum**) is formed of the median part of frontonasal process.
- **Lower lip** → formed by fusion of the two mandibular processes.

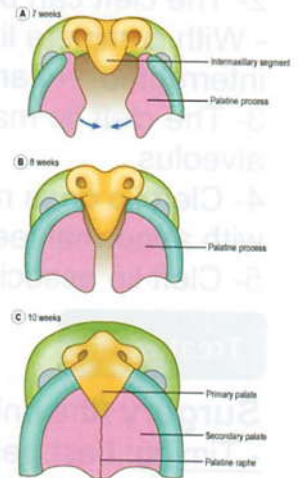
Development of the palate

A) Primary palate

- The anterior part of the palate & is also called the **premaxilla**
- It carries the four incisor teeth
- Derived from the median part of the frontonasal process.

B) Secondary palate

- From each maxillary process a palatal process grows medially across the dorsum of the tongue → the two palatal processes unite with each other & with the premaxilla together with the nasal septum → separate nasal cavities from each other & from the oral cavity



Congenital anomalies

- 1- **Cleft lip**
- 2- **Cleft palate**

The most frequent congenital anomalies of the face (1:700 live births)
 -The incidence is less in black & oriental races

3- Preauricular sinus

- Due to imperfect fusion of the tubercles that form the ear auricle
- When occluded → a cyst develop → might develop into abscess → bursts → resistant ulcer

4- Dermoid cysts.

- Sequestration dermoid cysts occur at the lines of embryonic fusion.
- The most frequent of which is the external angular dermoid

5- Pierre Robin syndrome consists of cleft palate associated with receding mandible (micrognathia) and posterior displacement of the tongue that is prone to obstruct the oropharyngeal airway.

6- Mandibular prognathism

- Definition: protrusion of the mandible.

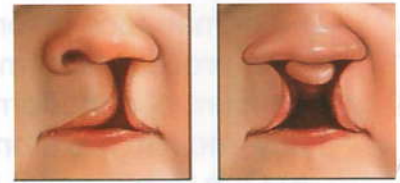
Cleft lip (harelip)

Etiology

- 1- Familial disease due to genetic susceptibility.
- 2- Consanguinity.
- 3- Prenatal exposure to alcohol, anticonvulsants, x-ray or virus infection as German measles in first 3 months of pregnancy

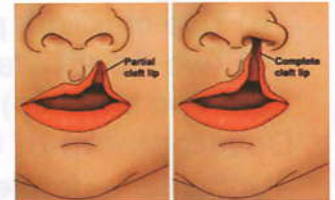
Pathology

- 1- Failure of fusion between the median part of the frontonasal process and one (unilateral) or both (bilateral) lateral maxillary processes in the developing face.
- 2- The cleft can be complete (reaching nostril floor) or incomplete.
 - With complete lip clefts → the orbicularis oris muscle is completely interrupted → flaring & flatness of the nares on the affected side
- 3- The cleft lip may be isolated or associated with cleft palate or alveolus
- 4- Cleft lip does not interfere with suckling, but it may be associated with abnormal teeth growth.
- 5- Cleft lip associated with other congenital anomalies in **35%**



Unilateral cleft lip

Bilateral cleft lip



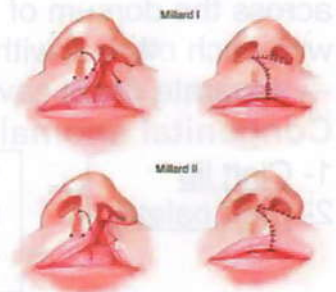
Treatment

Surgery (the only treatment)

- Timing: best performed at the age of **3-6 months**.

- Principles of the operation:

- 1- Paring of the edges.
- 2- Repairing the defect by suturing the three layers of the lip (skin, muscle and mucous membrane) taking care to adjust the vermilion and mucous borders.
- The sutures are not made in a straight line but in a zigzag way → avoid notching of the lip margin as the scar contracts



Cleft palate

Etiology

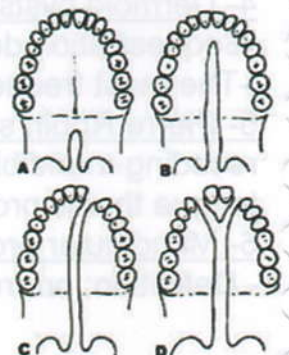
The same as for cleft lip

Pathology

- Arrest of fusion between two palatal processes with the premaxilla

Types

- A. Cleft soft palate.
- B. Cleft soft and hard palate (complete).
- C. Complete cleft palate plus one side of premaxilla (bipartite).
- D. Complete cleft palate plus both sides of premaxilla (tripartite)

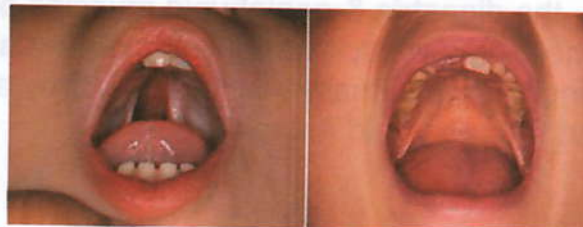
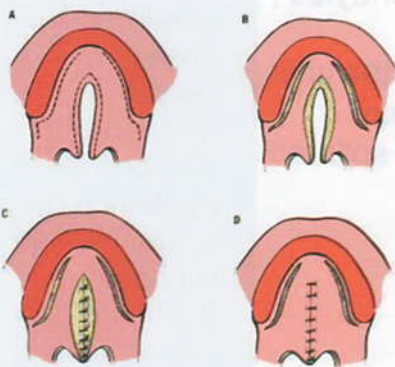


Effects on function

- 1- Inability to create a -ve intra-oral pressure (because of oro-nasal communication) → Impairment of normal suckling
- 2- Food will reflux into the nose → aspirated → aspiration pneumonia
- 3- Inadequate emptying of the middle ear (due to abnormal levator palate insertion) → prevents Eustachian tube adequate aeration → recurrent otitis media → Hearing loss
- 4- Speech defects secondary to:
 - a) Inadequate velopharyngeal mechanism (Opposition of velum "the muscles of soft palate" against the pharyngeal wall to separate the oral from the nasal cavity is impaired)
 - Nasal tone is due to naso-oral communication.
 - b) Hearing loss.
- 5- Distortion of facial growth.
- 6- Interference with normal teeth alignment.

Treatment

- Timing of operation: at **12-18 months**
- Preoperative management:
 - 1- Attention to feeding (In an upright position, feeds are given either by a bottle with a large hole or with a spoon)
 - 2- Prevention and treatment of chest infection.
- Objectives of surgery:
 - 1- Closure of oro-nasal communication
 - 2- Achieving a competent velopharyngeal sphincter
- Principles of surgery:
 - 1- Trimming of edges
 - 2- Suturing in 3 layers in middle line (nasal mucosa, muscle & oral mucosa)
 - 3- Lateral relaxation incisions are needed.
 - 4- Fracture of the pterygoid hamulus to relax the tensor palati.
- Post operative treatment:
 - 1- Speech therapy
 - 2- Orthodontic treatment



Maxillofacial Injuries

- Incidence: frequent with RTA, fights & contact sports
- The order of priorities in treatment of patients with extensive injuries:
 1. Patent airway (upper airway can be obstructed by blood, dentures or vomitus)
"Tracheostomy is rarely indicated"
 2. Effective breathing
 3. Control of haemorrhage by direct pressure or ligation of the bleeding vessels
- If patient is shocked → suspect associated abdominal or chest injuries
(Isolated facial injuries rarely cause shock)

Soft tissue injuries

Skin

- Minimal wound debridement.
- Cut wounds & lacerations → sutured
- Avulsed flaps → sutured after ensuring vascularity at the tip.
- If there are skin defects → local flaps or skin grafts

Lips

Sutured in 3 layers with respect to anatomical landmarks

Eyelid Injuries

- Careful suturing of all lid layers
- If cut levator not repaired → ptosis
- The tarsus should also be repaired

Ear Injuries

- Full thickness tears are sutured by cutaneous perichondrial sutures
- If haematomas not evacuated → cauliflower ear

Parotid injury

- Duct Injury → end to end anastomosis over a small silastic catheter. (Distal part of the duct → proximal cut end is sutured to the mucosa)
- Gland injury → skin is sutured & a small drain is inserted (there will be salivary leakage which usually stops in 3 weeks)

Nasal Injuries

- Nostril tears → sutured in 2 layers
- If septal haematoma is not evacuated → septal cartilage resorption → saddle nose deformity

Animal and human bites

- Usually heavily contaminated.
- Treatment: prompt excision, antibiotics & rabies vaccination for animal bites. The wound is left open.

Mandibular fractures

Site

- In the body (**the commonest**), symphysis, angle, ramus, condyles, coronoid or alveolar process.
- In bilateral cases, the digastric & geniohyoid muscles pull the chin fragment and attached tongue backwards → impair the airway
- Fractures of the angle are minimally displaced as they are splinted by the masseter and the pterygoid muscles.

- Fractures of the body usually occur close to the mental foramen where the bone is weakened by:
 1. The marked curvature
 2. The deep canine socket

Clinical picture

Symptoms

- 1- Pain, especially on attempts to open the mouth.
- 2- Blood-stained saliva.
- 3- Impairment of speech and swallowing.
- 4- Anaesthesia of the lower lip (Sometimes)

Signs

- 1- Swelling and haematoma in the floor of mouth.
- 2- Local tenderness
- 3- Crepitus
- 4- Irregularity of the line of teeth

Investigations

- 1- Plain X-ray (diagnostic)
- 2- A panoramic view shows the whole mandible
- 3- CT may be needed

Treatment

1. First-aid treatment

- Jaw is supported with 4-tailed bandage & give analgesics for pain
- Antibiotics and mouth hygiene are important to prevent infection

2. Reduction and fixation (done under anaesthesia)

- Indication: if there is displacement
- Fixation (for **3-6 weeks**) is done by:

- A. Arch bars
- B. Inter-dental wiring (the patient's jaws are wired together & given a liquid diet) → appropriate for the majority of lower jaw fractures.
- C. Plate and screws (for more complicated fractures)

Fractures of the mandible are usually compound into the mouth because the mucoperiosteum is firmly attached to the bone.



Fractures of the maxilla

Clinical picture

Pain, excess salivation, mal-occlusion, epistaxis, diplopia, swelling, and crepitations

Investigations

X-ray or CT scan

Classification

Le Fort classified

maxillary fractures into 3 types

- **Le Fort I:** a transverse fracture above the level of the teeth.
- **Le Fort II:** a pyramidal fracture, traversing the base of the nose through the posterior wall of maxillary antrum and across the orbit.
- **Le Fort III:** There is craniofacial disjunction, i.e., separation of the facial bones from their cranial attachment



Temporomandibular dislocation

- **Etiology:** 1- Direct blows 2- Yawning 3- Wide opening of the mouth under anaesthesia (commonly bilateral and affects middle aged females)

- **Clinical features:**

- Pain and dysarthria (the mouth is held open with fixed jaws)
- In unilateral cases, the chin is deviated to the opposite side.

- **Treatment:** Reduction (better under anaesthesia)

Method: downward traction on the molars with the padded thumb, with upward rotation of the body with the outside fingers.

Infections of the face

Dangerous area of the face

- **Definition:** The area of the face between the lines passing from the outer canthus to the angle of the mouth
- **Named so because:** infection in this area (as boils or carbuncles) is liable to cause cavernous sinus thrombosis
- **Route of spread:** ophthalmic veins or the pterygoid plexus → emissary veins (traverse the skull base) → to the cavernous sinus

Lip cancer

- **Etiology:** 1. Prolonged exposure to ultraviolet rays of the sun 2. Chronic smoking.

- **Pathology:**

A) Histological picture: Squamous cell carcinoma (the commonest) usually well differentiated

B) Gross picture

- **Site:** The lower lip is more affected than the upper.
- The lesion usually starts as a nodule or erosion which resists treatment. Later the typical ulcer becomes evident:
 1. Raised everted edges
 2. Indurated base and margin (induration extends beyond the edge)
 3. Possibly spread to cervical LNs:

- I) The central part of lower lip → submental nodes
- ii) The lateral parts → submandibular nodes

Upper deep cervical LNs

- **Treatment:**

- **1st tumour** → surgical excision of the lesion with a safety margin or by radiotherapy (squamous cell carcinoma is radio-sensitive)

- **Lymph nodes.** If there are LNs metastases → suprahyoid or a complete block dissection

- **Prognosis:**

- Epitheliomas of the lip have better prognosis than those in the rest of the oral cavity

Nose Fractures

- **C/P:** pain, swelling, epistaxis & crepitus

- **Investigation:**

- Plain X-ray

- **Treatment:**

- reduce the fracture by hand or using an instrument (fix the position by intranasal packing for **3 days** with an external splint for **7 days**)

Zygoma Fractures

- **C/P:** pain, swelling of eyelids, flat cheek, crepitus & numbness of cheek & upper teeth (due to infraorbital nerve injury) with irregular infraorbital margin

Blowout fractures

- **Definition:**

- orbital floor depressed fractures with herniation of some of orbital contents into maxillary sinus

- **Cause:** sudden increase in the intraorbital pressure.

- **C/P:** diplopia and limited up and down gaze, with or without enophthalmos

Mouth , cheek and tongue

Stomatitis

Predisposing factors

1. Anaemia and VB 12 deficiency → thin, atrophic epithelia and loss of tongue papillae
2. Immunodeficiency and autoimmune diseases.

Types

1. Aphthous stomatitis

- Painful ulcers up to **0.5 cm** across
- Round or oval with a yellow base and red margin
- They heal normally within **10-14 days.**

2. Herpes simplex infections.

- Small vesicles appear and rapidly break down to form small, yellow ulcers with bright red margins.

-Site:

Gingiva, cheeks, lips, tongue and skin of the cheek & around nostrils

- The patient is unwell, febrile with enlarged submandibular LNs

3. Monilial stomatitis

- This is a fungus infection by candida species
- C/P: formation of white membranous lesions.
- Type of patient: debilitated, chronically ill patients under cytotoxic drugs, cortisone therapy or prolonged broad –spectrum antibiotics
- It affects mainly the tongue
- Treatment: by gentian violet and amphotericin-B lozenges.



Cysts of the floor of mouth

1- Mucous cysts of minor salivary glands

- Either retention cysts behind minute calculi or extravasation cysts
- They form soft pinkish or bluish swellings up to 1.5cm in diameter
- Treatment: excision

Ranula

Definition

A retention cyst arising from a sublingual salivary gland
(Saliva distends cyst in the floor of the mouth)

Pathology

- Wall is composed of a thin fibrous capsule lined by macrophages
- It may contain gelatinous material.
- If it extends down into the neck over posterior margin of the mylohyoid (diaphragm of the mouth) → a plunging ranula
- It may rupture but usually refills again.

Clinical picture

Translucent, Bluish, cystic swelling with prominent blood vessels running over its surface with stretched submandibular duct

Treatment

- Excision of the sublingual gland with the cyst is very difficult (as the wall is thin and adherent).
- Marsupialisation (Partial excision of the roof then suturing the edges to the mucosal lining the floor of the) → treatment of choice.

2- Lingual and sublingual dermoid

- C/P: Opaque swellings lined by stratified squamous epithelium.
- Pathology: The cysts are filled with mucous (fluctuant) or with a doughy mass of keratin.
- Site: They are found in the midline of the tongue or in the floor of the mouth (either in midline "the commonest" or laterally in the submandibular region)
- Presentation: after puberty (although congenital)
- Treatment: excision through the floor of the mouth (If large → submandibular incision)



Developmental anomalies of the tongue

Macroglossia

-Definition: Persistent, painless enlargement of the tongue.

- Causes:

A) Congenital causes

1. Cavernous haemangioma
2. Congenital arteriovenous fistula
3. Lymphangioma
4. Neurofibromatosis

B) Acquired causes:

1. Cretinism
2. Acromegaly
3. Amyloidosis



Macroglossia (abnormally large tongue)



ADAM



Tongue tie

- Etiology: short fibrous lingual frenulum

-Effect: impairment of tongue movements & speech defects

-Treatment: division of the frenulum below the undersurface of the tongue.

Congenital fissured tongue

Fissures run transversely from a median groove (unlike those of syphilis, which are longitudinal)

Inflammation of the Tongue (glossitis)

Chronic superficial glossitis (is precancerous)

Etiology

Chronic irritation (as smoking, sharp tooth, sepsis, syphilis, spirits & spices)

- It occurs in middle and old ages.

Pathology

- There will be hypertrophied papillae, leukoplakia, red glazed tongue, cracks, fissures and warty projections.
- Hypertrophy of the papillae produces red hyperemic patches
- Overgrowth of the epithelium makes it thickened, indurated and opaque so that the red patches are replaced by white ones & tongue appears as if it were coated with white paint (**leukoplakia**)
- In the next stage, epithelium may be shed over a considerable area → a red glazed tongue.
- Submucous fibrosis occur → ulcers, fissures & warty projections

Treatment

1. Elimination of the irritating source
2. Mouthwash
3. In resistant cases → excision of lesion



Tongue Ulcers

1. Dental ulcer

- Etiology: repeated trauma by a broken tooth or ill fitted denture
- Site: at the side of the tongue near the offending tooth (might not be apparent when the tongue is protruded for inspection)
- Gross picture: The ulcer is small, oval or rounded with granulation tissues at the floor, soft base and sloping margins.
- Clinical picture: painful with septic enlargement of draining LNs
- In chronic cases the edges may be raised with indurated base → biopsy is needed to exclude malignancy
- Treatment: 1. removal of the offending cause
2. The use of an antiseptic mouthwash.

2. Aphthous ulcers

- Etiology: not exactly known
- Pathology: multiple, small ulcers affecting all mucosal lining of the oral cavity with red margin & soft base
- C/P: very painful, rounded, yellow ulcers & no LN enlargement
- Site: near the tip of the tongue
- Treatment: (normally heal in **2 weeks**)
By antiseptic mouth wash, alkaline lotion as 2% sodium bicarbonate and anaesthetic gel

Tongue Injuries

- Etiology

1. Biting the tongue (the commonest)
As in epileptic patients during attacks
- 2 With jaw fractures due to RTA

- Complication:

Damage to the lingual artery → bleeding → endanger the airway in unconscious patients

- Treatment:

1. Arrest bleeding by hooking the tongue forwards with a finger & compressing it against & mandible
2. Then suture the lacerations in the OR

3. Neoplastic ulcers

Usually squamous cell carcinoma

Carcinoma of the Tongue

Incidence

- Usually above 60 years.
- Nowadays, it is just slightly higher in males
 - The recent lowering of the incidence in males is due to:
 - a) More efficient treatment of syphilis
 - b) Improved oral hygiene
 - c) Discontinuing use of clay pipes
 - d) Reduced spirits consumption
 - Recent increased females incidence is due to increase of smoking

Etiology

A) Predisposing factors: Chronic irritation by smoking, sepsis, spices, spirits, sharp teeth & bad oral hygiene

B) Precancerous lesions

- 1- Dental ulcers
- 2- Chronic superficial glossitis
- 3- Leukoplakia (any white lesion which cannot be rubbed off)

Pathology

Site

- 1- The lateral margin of the anterior 2/3 (The commonest, 50%)
- 2- The posterior 1/3 (the 2nd common)
- 3- The ventral & dorsal surface
- 4- The tip (rare)

Gross types

1. Malignant ulcer with deep irregular floor with necrotic tissues raised nodular everted edges and a hard indurated base.
 2. Raised oval plaque
 3. Diffuse infiltrating tumour (**wooden base**) is rare.
- The surrounding mucous membrane may show leukoplakia.

Microscopic appearance

- 1- Squamous cell carcinoma (90%)
- Tumours of the posterior third are usually less differentiated

Spread

1. Direct spread

- Tumours of anterior 2/3 → laterally and may reach the floor of the mouth before they cross the midline (may invade the mandible)
- Tumours of posterior 1/3 → to tonsils, pharyngeal wall and larynx. (May cross to the other side of the tongue)

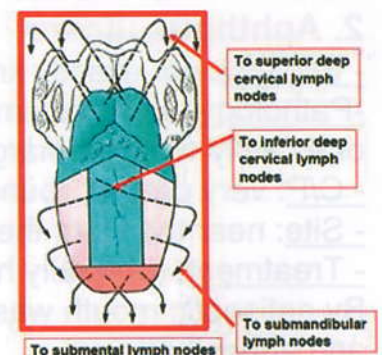
2. Blood spread (rare, more in tumours of the posterior third)

3. Lymphatic spread (occurs early)

- Of the tip of the tongue → submental LN → bilaterally to submandibular → upper deep cervical nodes
- Of the lateral 1/3 → ipsilateral submandibular → upper deep cervical LNs (Those near the midline disseminate bilaterally)
- Posterior third → directly to the upper deep cervical LNs



Carcinoma in situ, means evidence of malignant cells without invasion of the basement membrane → no lymphatic or blood spread



Complications

1. Inhalation of necrotic tissues → bronchopneumonia
2. Combined cancer cachexia & starvation due to pain and dysphagia.
3. Haemorrhage from lingual artery erosion
4. Asphyxia due to either pressure of enlarged fixed LNs on the air passages or due to edema of the glottis

Clinical picture

A) Early cases

Symptomless (patient may complain of a persistent ulcer with indurated base and everted edges)

B) Late presentation

1- Pain in tongue

- Cause: 1st due to infection, later due to lingual nerve infiltration.
- Referral: to the ear or temple through the chorda tympani via the auriculo-temporal nerve
- In tumours of the posterior third pain may be felt on swallowing.

2- Salivation due to the pain plus restricted tongue mobility.

- It may be blood stained & smells badly (necrosis and infection).

3- Inability to articulate clearly.

4- Enlarged cervical lymph nodes.

5- Ankyloglossia (tongue fixation)

Due to extensive infiltration of the floor of mouth

Investigations

1. Biopsy from the ulcer edge
2. Fine needle aspiration cytology of suspected cervical LNs
3. CT of neck and mandible.

Treatment

Radical treatment for early cases

A) Surgery

- Indications:

1. Small growths (T1 and T2)
2. Incomplete regression or recurrence after radiotherapy.
3. Cancer on top of a precancerous lesion as leukoplakia.
4. Presence of the tumour very close to the mandible or infiltrating it.

- Preoperative preparation:

- 1- Care of teeth and oral hygiene.
- 2- Preoperative irradiation by 4000 rad may be advised.

TNM-staging

To	No evidence of tumour
T _{is}	Carcinoma in situ
T1	<2 cm
T2	2-4 cm
T3	>4 cm
T4	Involvement of base
No	No lymph nodes
N1	Ipsilateral single node <3 cm
N2	Ipsilateral or contralateral LNs <6 cm
N3	Lymph nodes >6 cm
Mo	No metastasis
M1	Distant metastasis

-The classic picture of tongue cancer is that of an old man sitting in the outpatient clinic with cotton wool in his ear & blood stained saliva dribbling from the mouth.
- Any ulcer or fissure-like lesion that resists treatment should be biopsied

Surgery and radiotherapy are the main lines of treatment. Chemotherapy is used as an adjuvant in some cases.

- Resection procedures

- 1- Excision with a safety margin of **1.5-cm** → partial or near total glossectomy
- 2- If there are metastatic LNs → modified neck dissection
- 3- If the mandible is affected → excised together with the tongue and the affected LNs (Combined mandibulectomy and neck dissection operation is known as **Commando operation**)
- 4- Close the huge defects following major resectional operations on tongue, floor of the mouth & mandible by various plastic procedures as pectoralis major myocutaneous flap

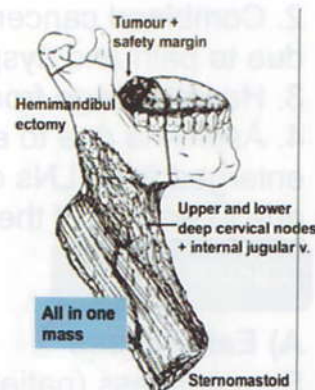
B) Radiotherapy

- Indications:

T1 and T2 (**< 4 cm**) "equally benefit from surgery or radiotherapy"

- Advantages: Avoiding the disfiguring side effects of surgery

- Disadvantages: Mucositis, dysphagia and osteoradionecrosis



Carcinoma of the Cheek

Etiology

(As cancer tongue)

A) Predisposing factors: Chronic irritation by smoking, sepsis, spices, spirits, sharp teeth & bad oral hygiene

B) Precancerous lesions

- 1- Dental ulcers
- 2- Chronic superficial glossitis
- 3- Leukoplakia (any white lesion which cannot be rubbed off)

Pathology

- 1- Squamous cell carcinoma (**the commonest**)
- 2- Adenocarcinoma of minor salivary glands (less common)

Clinical picture

An ulcerated mass with raised everted edges & indurated nodular floor

Treatment

- 1- Interstitial or external beam radiation (treatment of choice)
- 2- Surgery

- Indication: recurrent or residual tumors

- Followed by reconstruction (as for the tongue cancer)

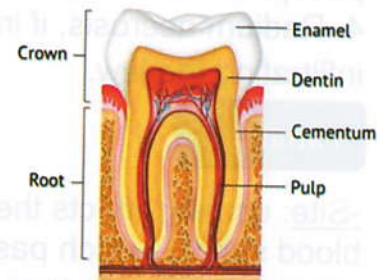
- Lymph nodes metastasis are treated by neck dissection



Teeth , gums and jaws

Structure of the Tooth

- Consists of 2 parts: a) The crown → projecting beyond the gum
b) The root embedded in the alveolus of the bone
- It has a central cavity (called **the pulp**) which is:
 - a) Rich in blood vessels and nerves.
 - b) Surrounded by the dentine (similar to bone but contains no cells)
- The enamel covers the crown while the cementine covers the dentine of the root.
- The cementine is surrounded by the dental periosteum (Periodontal membrane)
- The gum is formed of mucous membrane that is firmly attached to the periosteum at the alveolar margin of the bone



Alveolar abscess

Etiology

Spread of infection from necrotic pulp into the periapical tissue.

Complication

Pus burst under periosteum → subperiosteal abscess → points externally or in maxillary antrum → infection spread to cavernous sinus

Clinical picture

- 1- Marked toxæmia
- 2- Severe pain and cheek swelling with gum inflammation.
- 3- Regional lymph nodes are enlarged and tender

Treatment

- 1- Antibiotics
- 2- Extraction of the offending tooth to provide drainage
- 3- Incision and drainage of the subperiosteal abscess

Osteomyelitis

Acute osteomyelitis (usually turns into chronic)

- **Etiology:** affects lower jaw as a complication of an alveolar abscess

- The commonest organism is **Staphylococcus aureus**.

- **Clinical features:**

- 1- History of alveolar abscess followed by pain, fever and swelling
- 2- Trismus may be present.

- **Treatment:** Antibiotics and mouth wash

Chronic osteomyelitis

Etiology

1. Following acute osteomyelitis.
2. As a complication of a compound fracture of the mandible.
3. Chemical necrosis due to substances used in industry, e.g., phosphorus, arsenic and mercury.
4. Radium necrosis, if irradiation is used for tumours nearby or infiltrating the jaw.

Pathology

- Site: usually affects the lower jaw because it has a single arterial blood supply, which passes near and parallel to the edge (in the upper jaw vessels pass at right angles to the jaw)
- Infection starts in medulla & spreads to the subperiosteal space.
- It is similar to osteomyelitis in other bones, differences include:
 - 1- Involucrum is poor.
 - 2- Sequestra take a long time to separate.
 - 3- If inferior dental artery is thrombosed → massive sequestrum

Clinical picture

- Pain, fever & jaw thickening → followed by abscess & sinus formation
- If the molar region is involved → Trismus

Investigations

1. Plain X-ray → bone necrosis (sequestrum) and new bone formation (characteristic).
2. Culture and sensitivity of the discharged pus.
3. CT scan.

Treatment

1. Antibiotics and mouth hygiene.
2. Incision and drainage of an abscess, if present.
3. Saucerization and Sequestrectomy through an incision over the lower border of the mandible.



Classification of Jaw Swellings

A) Epulides = swellings of muco-periosteum or gum margin

1. Fibrous epulis

2. Granulomatous epulis
3. Myeloid epulis.

4. Sarcomatous epulis.

5. Carcinomatous epulis.

B) Odontomes = cysts or cystic tumours related to development of the teeth:

1. Dental cyst.
2. Dentigerous cyst.
- 3.

Adamantinoma (ameloblastoma)

C) Bone Tumours:

I. Benign

1. Osteoma.
2. Chondroma
3. Giant cell lesions of the jaw

II. Malignant

1. Osteogenic sarcoma.
2. Fibrosarcoma.
3. Secondaries.
4. Malignant tumours of the maxilla.

Epulides

Definition

An epulis is a solid swelling rising over the gum

Fibrous epulis

- Definition: localized inflammatory hyperplasia of gum submucosa
- Etiology: chronic irritation
- Clinical picture: a small, painless, pedunculated swelling arising between two teeth.
- Treatment: extracting the teeth on either side of the tumour and excision of the tumour with a wide base of mucoperiosteum.



Granulomatous epulis

- Definition: a mass of granulation tissue adjacent to a carious tooth or gingivitis.
- Pathology: It forms a red, soft, lobulated mass over the jaw, which bleeds easily on touch because it is devoid of epithelial cover.
- Treatment: Excision of the excess granulation tissue is done with diathermy, together with removal of the offending tooth



Myeloid Epulis

- Incidence: affects the lower jaw more than the upper.
- Origin: from inner osteoclastic layer of periosteum or alveolus itself
- Grossly: a soft, lobulated, encapsulated swelling with cystic areas in cut sections
- Microscopically: spindle cells & giant cells together with fibroblasts and fibrous tissue.
- Clinical features: The swelling is sessile, lobulated, bluish or brownish & covered with intact mucosa (fixed to underlying bone)
- As it enlarges, the adjacent teeth are loosened and fall off.
- The mucous membrane is intact unless bitten by the upper teeth.
- Investigation: X-ray of the jaw → shows eating up of the bones.
- Treatment: Wide excision with a safety margin



Sarcomatous epulis

- A Parosteal fibrosarcoma arising from outer layer of periosteum
- Clinical features: The tumour is sessile and fixed to the bone. It is of variable consistency.
- Investigation: X-ray → shows infiltration of the bones
- Treatment:



Wide excision with the underlying bone up to hemimandibulectomy

Carcinomatous epulis

- Definition: an epithelioma arising from the mucous membrane covering the gum.
- Clinical picture & treatment: similar to carcinoma of the floor of mouth, but radiotherapy has no role

Odontomes

Definition

Cysts or cystic tumours related to the development of teeth

Types

	Dental cyst	Dentigerous cyst
Pathogenesis	Develops in connection with a pulpless infected tooth, which causes chronic irritation of the paradental epithelial debris of Malassez	At or after 2 nd dentition in relation to an unerupted tooth (canine, premolar or third molar). - It affects the lower jaw more commonly -It may be due to irritation of paradental epithelial debris of Malassez or cystic degeneration of dental follicles.
Type of patient	Usually occurs in adult	In children and adolescents
C/P	1. a painless, slowly growing swelling that usually arise in the anterior part of upper jaw 2. As it expands the jaw, the bone becomes so thin that it gives a sensation of egg shell crackling when pressing on it	• This is similar to dental cyst, but it usually occurs in the lower jaw near the angle of the mandible and there is a missing tooth (unerupted)
Investigation	X-ray → expansion of the jaw around a well defined cavity containing no teeth or trabeculae	X-ray → expanded jaw and a clear cyst with a tooth inside it
Treatment	- Extraction of the affected tooth then open the cyst through an incision in the gum & enucleation of the cyst with removal of the lining membrane & fluid - The bone may be crushed to keep the shape of the alveolus 	- Deroofing of the cyst with removal of the lining epithelium and fluid. -The unerupted tooth is removed. - The expanded jaw is crushed to restore its shape 

Adamantinoma (ameloblastoma)

Incidence

- Affects middle aged females (25-45 years).
- It occurs commonly in the **lower** than the upper jaw

Pathology

- Adamantinoma is a locally malignant tumour.
- **Starts** near the **angle** of the mandible and grows slowly both
 - a) Forwards → in body of the mandible
 - b) Upwards → in the ascending ramus.
- It is well encapsulated by fibrous tissue and expands the jaw.
- Trabeculae traverse the tumour dividing it into lobes and lobules.
- The cut section is pink or whitish with multiple cystic & solid areas.
 - A) **Cystic** areas are lined by columnar cells & contain brownish mucoid fluid
 - B) **Solid** areas consist of fibrous tissue with particles of enamel
- Osteoclasts are not present.

Clinical picture

- A painless, lobulated, slowly growing swelling (usually in lower jaw)
- It expands the jaw more on the outer than the inner side → more obvious from the cheek than from the mouth.
- It is well defined and not tender.
- The swelling is bony hard in consistency, but egg shell crackling can be elicited over large tumours

Investigations

- 1- Plain X-ray → an expanding, translucent, well defined shadow, divided by bony septa into a more or less equalized lobules (fine soap bubble or honey comb appearance)
2. CT scan and MRI.
3. Biopsy.

Treatment

- 1- Resection of the part of the mandible carrying the tumour with a safety margin (which may amount to hemimandibulectomy)
- 2- Reconstruction done either immediately or at 2nd stage by a bone graft (free or vascularized rib graft) or by prosthesis



Complications

1. Ulceration, infection, bleeding and falling of teeth.
2. It is locally malignant → liable to recurrence after inappropriate surgery.
3. Sometimes, it turns malignant, (either from fibrous tissue element → sarcoma or from epithelial cells → carcinoma)



BONE TUMOURS OF THE JAWS

A) Benign tumours and tumour-like conditions

Osteoma

- May be of an ivory or a cancellous variety:

I- Ivory osteoma affects the bones which develop in membrane as the ascending ramus of the mandible, outer surface of the maxilla, palate, antrum and orbit.

- Treatment by excision

II- Cancellous osteoma affects the bones which develop from cartilage as the maxilla & the symphysis menti

- Treatment is by excision

Giant cell lesions of the jaw

I-Giant cell granuloma

Pathology

-Site: affects the lower jaw more commonly

-Gross: This is like osteoclastoma in large bones, but is benign.

- It arises above symphysis menti as a soft reddish mass & grows backwards in body of mandible but not upwards in ascending ramus.

-The cut section shows a lobulated tumour.

- Histologically: giant cells, spindle cells & numerous blood vessels.

- It is similar to brown tumours of hyper-parathyroidism and to giant cell epulis (**myeloid epulis**).

Clinical features.

A painless, slowly growing tumour, expanding the jaw equally on both sides (due to presence of osteoclasts) → egg shell crackling

Investigations:

1- Plain x-ray → expanding translucent shadow with unequal trabeculae (soap bubble appearance).

2- CT scan and MRI.

3- Biopsy

4- Serum calcium & parathormone to exclude hyperparathyroidism.

Treatment:

1- Careful enucleation and gentle curettage.

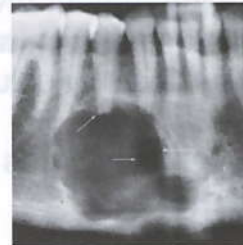
2-In large tumors → resection of the mandible

II- Osteoclastoma:

This is a rare tumour that is similar to the foregoing, but is malignant on histological examination.

III- Aneurysmal bone cyst:

It forms a soft, sponge like tumour which oozes blood until enucleated completely.



B) Malignant bone tumors

1. Osteogenic sarcoma

- Commoner in the upper jaw.

-May occur on top of Paget's disease of bones & is similar to osteogenic sarcoma

2.

Fibrosarcoma

(parosteal fibrosarcoma).

- May arise from the jaw periosteum

3. Secondary carcinoma.

-Reach the jaw by direct spread from nearby carcinoma as lip, tongue or floor of mouth.

-Treatment: is that of the primary together with resection of the mandible (no role for radiotherapy to avoid mandible necrosis)

4. Malignant maxillary tumours:

i) Carcinoma

ii) Sarcoma:

1. Osteogenic sarcoma

2. Spread from ethmoid sarcoma.

3. Malignant lymphoma

(**Burkitt's lymphoma**).

Carcinoma of the maxilla

Incidence

- The commonest malignant tumour of maxilla.
- It affects both sexes equally
- Usually occurs at about the age of **40 years**

Clinical picture

I. In early cases

- Suspected if: a) pain & chronic sinusitis in an old patient fail to respond to the ordinary treatment.
- b) Bleeding occurs with every puncture
- c) There is deep pain in antrum.

II. Presentation depends on infiltrated wall of maxillary antrum

- a) Anterolateral wall infiltration → bulging & swelling of the cheek
- b) Medial wall infiltration → unilateral foul nasal discharge (mucous, blood and pus) due to nasal obstruction. Later on, obliteration of the nasolacrimal duct → epiphora
- c) Posterior wall infiltration → invade the nasopharynx → change of voice, difficulty of breathing & post-nasal discharge of pus & blood
- d) Roof (floor of the orbit) infiltration → proptosis and diplopia.
- e) Floor of the antrum (the hard palate) infiltration → depression & bulge of the palate into the mouth.

III- In late case, patient may present with 2^{ry} LN enlargement

Investigations

1. Plain X-ray

- In early stages → opacity and increase in size of the antrum.
- Later → decalcification & erosion of bone.

2. CT scan & MRI → define the exact site, size and extent of tumour

3. Maxillary sinus endoscopy (sinuscopy) & biopsy.

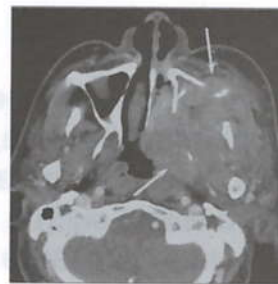
Treatment

I-Early cases

- Both surgery and radiotherapy produce equal results.
- Surgical treatment is recommended in cases with bone invasion as curability with radiotherapy is reduced in these patients.
- The standard operation → **total maxillectomy** (the whole maxillary antrum is removed with its walls then a synthetic prosthesis is fitted in place of the excised bone to avoid facial deformity)
- If cervical LNs are involved → Radical block neck dissection

II- Advanced cases

- A combination of radiotherapy & surgery is better than either alone.
- After a course of external irradiation, maxillectomy is performed.



Predisposing factor

- Chronic infection of the antrum (in most cases it is associated with 2^{ry} infection of the nasal sinuses)

Pathology

- Origin: arises from the antrum and encroaches on one or more of its walls and involves the surrounding structures.

- Histologically: may be columnar or squamous cell carcinoma.

- Spread:

a) Direct → by infiltration of adjacent structures

B) Lymphatic

→ to the upper deep cervical & retropharyngeal LNs

C) Blood borne (rare)

The Neck

Anatomical divisions of the neck

A) Triangles

- Each side of the neck is divided by sternomastoid muscle into:

1- Posterior triangle.

2- Anterior triangle → further divided into:

a) Submandibular triangle, whose boundaries are:

- i- Above → body of the mandible
- ii- Posteriorly → posterior belly of digastric
- iii- Anteriorly → anterior belly of digastric

b) Carotid triangle, whose boundaries are:

- i- Posteriorly → sternomastoid muscle
- ii- Superiorly → posterior belly of the digastric
- iii- Inferiorly & anteriorly → superior belly of omohyoid

c) Muscular triangle, whose boundaries are:

- i- Posteriorly → the sternomastoid muscle
- ii- Superiorly → the superior belly of omohyoid
- iii- Anteriorly → the middle line.

(The two muscular triangles form together a diamond shaped area)

B) Compartments

In a cut section, the neck is divided into two compartments.

1- The musculo-skeletal compartment

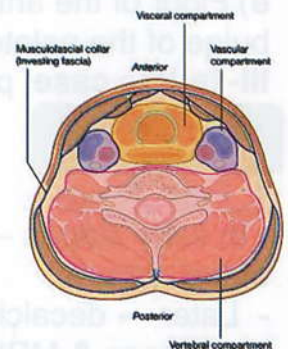
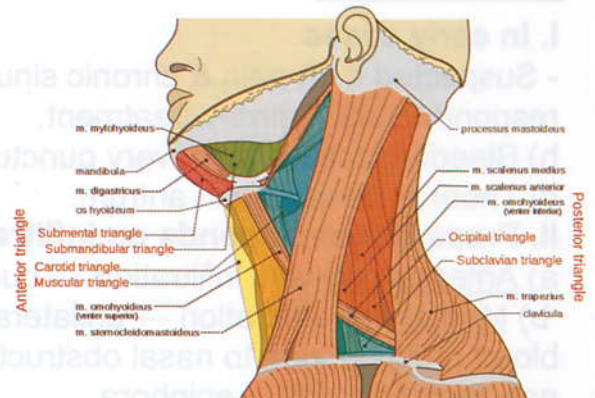
- Lies **posteriorly** & is separated from the visceral compartment by thick fascia (**the prevertebral fascia**)

2- The visceral compartment

- Lies **anteriorly** and is enclosed in the investing fascia of neck.

- **Contents:** the oesophagus, trachea, thyroid & parathyroid glands, carotid arteries, vagus nerve and the jugular veins.

- Because it is enclosed within such a fascial covering a swelling in this visceral compartment → pressure symptoms & signs as dysphagia, dyspnoea or weak carotid pulse



Neck injuries

Etiology

A) **Penetrating injuries** (more common) → by gunshots or stabs

B) **Blunt injuries** Usually 2nd to RTA & may cause spine fracture

Neck injuries are serious because

1. The neck contains vital viscera and vessels.
 2. Unstable cervical spine fractures & dislocations can cause paraplegia, Quadriplegia or immediate death
 3. Neck injuries are commonly associated with head, face & chest injuries
- The most commonly injured structures are in that order: the big vessels, spinal cord and the aerodigestive tract

Clinical picture

A) Vascular injuries present by

- 1- Hemorrhage (may be severe enough to cause shock)
- 2- Expanding hematoma.
- 3- Injury to a large vein may lead to pulmonary embolism.

B) Neurological injuries

- 1- Fracture of the cervical spine → quadriplegia or paraplegia.
- 2- Injury of the brachial plexus or cranial nerves.

C) Injuries of the larynx or trachea may present by respiratory obstruction, aphonia, hoarseness of voice, subcutaneous emphysema, air bubbling through the wound or hemoptysis

D) Esophageal injuries → leakage and severe infection in the neck
→ spread to the mediastinum

Investigations

1- Plain X-ray may reveal:

- a) FB b) Wide mediastinum c) S.C. emphysema d) Fractures

2- Duplex ultrasound → injury to the big vessels.

3- CT of the skull and brain

4- CT angiography

5- For laryngotracheal injuries → Laryngoscopy and bronchoscopy.

6- For esophageal injuries → Gastrografin swallow

Treatment

I- ABCDE of trauma must be followed

- 1- Airway management especially endotracheal intubation can be both difficult & dangerous (done by the most experienced personnel)
- 2- Cricothyroidotomy may be easier and safer than tracheostomy
- 3- Trendelenburgh position → avoid air embolism in major venous injuries
- 4- IV lines should be avoided in the side of trauma.

II- Tension pneumothorax

- Managed by inserting a needle in the 2nd intercostal space.

III- External bleeding

- a) Minor → local compression
- b) Major → exploration to stop bleeding & perform repair

IV- In case of neurologic damage

- Neck support & evaluation of patient initially according to Glasgow coma scale then by CT and/or MRI to define the neurological lesion

V- Esophageal injury

- If discovered early → immediate exploration and repair

VI- Laryngotracheal injuries → surgical repair

Cellulitis of the neck

I- Superficial cellulitis

Common and is diagnosed & treated as cellulitis anywhere

II- Deep cellulites (Ludwig's angina)

Pathology

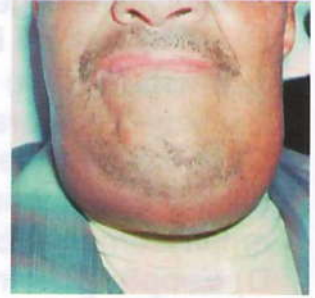
- Inflammatory swelling of both submandibular region & mouth cavity
- It is due to deep cellulitis around the submandibular gland usually due to streptococcal infection (deep to the deep fascia) → spread to the glottis → edema and asphyxia.

Clinical picture

- 1- An inflammatory swelling in the submandibular region that is painful, red, warm, tender & overlying skin is edematous
- 2- Swelling of the tongue which is pushed upwards and forwards

Treatment

Urgent antibiotics and drainage through an incision beneath the jaw (incision deepened to permit displacement of submandibular salivary gland & division of mylohyoid muscle → adequate drainage)



Cervical lymph-adenopathy

½ of the LNs of the body are present in the neck → drain the head & neck.

-In addition, the supraclavicular nodes are 2nd stations for the apex of lung, upper limbs, abdominal viscera and testes → Accordingly, cervical lymph-adenopathy is very common.

Differential diagnosis of neck swellings

I- Mid-line swellings

A) Solid swellings

- 1- Submental LN enlargement.
- 2- Nodule in the isthmus of the thyroid gland.

B) Cystic swellings

- 1- Dermoid cyst.
- 2- Thyroglossal cyst (moves up & down with deglutition also moves up with tongue protrusion)
- 3- Subhyoid bursitis
-A rare tender, oval swelling which lies transversely below the hyoid
-It moves up & down with deglutition and with tongue protrusion
- 4- Cysts in the thyroid gland.
- 5- Laryngocoele
- Rare swelling in musicians who play air-blown instruments, it is herniation of laryngeal mucosa through thyrohyoid membrane
- It is compressible & increases in size with coughing or blowing

II-Swellings in the submandibular triangle

- 1- Enlarged submandibular LNs (multiple and can be rolled over the edge of the mandible)
- 2- Enlarged submandibular salivary gland

III- Swellings in the carotid triangle

A) Solid swellings

- 1- Enlarged upper deep cervical LNs
- 2- Carotid body tumour

B) Cystic swellings

- 1- Cold abscess
- 2- Branchial cyst

IV-Swellings in the posterior triangle

A) Solid swellings

- 1- Enlarged LNs
- 2- Cervical rib
- 3- Neurofibroma arising from the brachial plexus.

B) Cystic swellings

- 1- Cystic hygroma
- 2- Pharyngeal pouch
- 3- Cold abscess.
- 4- Pneumatocoele → caused by herniation of the pleura into the base of the neck → a cystic swelling in the supraclavicular region (Resonant and compressible)

V-Other swellings that may arise anywhere

- Swellings of skin & S.C. tissue are common in the neck
- They are added to any of the previous lists.

- 1- Lipomas
- 2- Sebaceous cysts
- 3- Haemangiomas

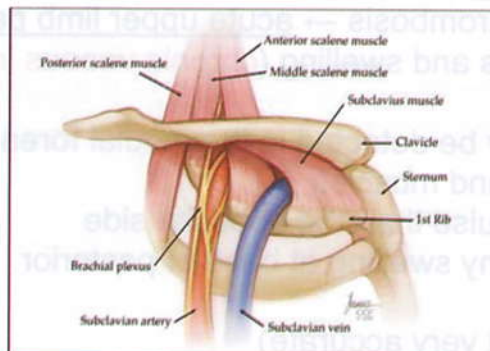
Thoracic outlet syndrome

Anatomy

- The brachial plexus & subclavian artery pass to upper limb through a narrow triangle in the base of the neck
- This triangle is bounded:
 - a) Anteriorly → scalenus anterior muscle
 - b) Posteriorly → scalenus medius muscle
 - c) Inferiorly → the first rib
- At this narrow space compression of the nerves & artery occur→ symptoms and development of thoracic outlet syndrome

Etiology

- 1- Cervical rib (complete or incomplete) → bony structure extends from the 7th cervical vertebra to the 1st rib.
- 2- A fibrous band extending from an incomplete cervical rib which ends at the first rib.
- 3- A tight scalene muscle.
- 4- Post fixation of the brachial plexus where the lower root of the brachial plexus arises from T2 instead of T1 →T2 becomes excessively stretched over the first rib



Carotid body tumour

- Incidence: a rare slowly growing malignant tumour
- Origin: from chemoreceptors at bifurcation of carotid artery.
- C/P: 1. slowly growing swelling at middle age which is usually smooth but may be lobular
- 2. Swelling moves from side to side but not vertically.
- 3. Exhibits transmitted pulsations from underlying carotid artery.
- Investigation: Angiography → splaying of the Bifurcation
- Biopsy even with a fine needle is contraindicated as tumour is highly vascular.
- Treatment: is excision with preservation of the internal carotid artery.
- If not possible to separate from the tumour, the ICA is excised & replaced by a synthetic graft

I- Brachial plexus

- The lower trunk is the main structure to be compressed
- It contains: a) sensory fibers to medial aspect of hand & forearm
b) Motor fibres to small muscles of the hand.
c) Most of the sympathetic fibers of the upper limb.

II- Subclavian artery (Less common to be compressed)

- Leads to: 1- Chronic ischaemia of the upper limb.
- 2- Post-stenotic dilatation of the artery segment immediately following the compression.
- 3- Post-stenotic dilatation → a thrombus inside → may send a shower → emboli to index & middle fingers (are direct continuation of brachial artery) → digital gangrene.

III- Subclavian vein

- Its Compression leads to: 1- Subclavian & axillary vein thrombosis
- 2- Chronic venous insufficiency of the upper limb

Clinical picture

Type of patient

- More frequently in females
- Rarely develop before adulthood.
- Usually unilateral affecting dominant arm but sometimes bilateral

Symptoms

1- Nerve compression symptoms (the commonest):

Tingling & numbness in the medial aspect of the forearm & hand due to compression on the lower root of the brachial plexus

2- Arterial symptoms:

- a) Intermittent claudication pain in upper limb (with exercise)
- b) Raynaud's phenomenon (occurs in cold weather)

Attacks of pallor in fingers followed by cyanosis then redness (**rebound hyperaemia**) due to irritation of sympathetic fibres supplying upper limb

3- Venous symptoms (rare)

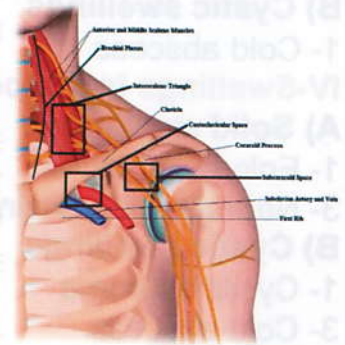
- a) Subclavian vein thrombosis → **acute upper limb pain & swelling**
- b) Chronic heaviness and swelling (**chronic venous insufficiency**).

Signs

- 1- Hypoesthesia (may be detected in the medial forearm & hand)
- 2- Mild wasting of hand muscles
- 3- A weaker radial pulse than the opposite side
- 4- Cervical rib → bony swelling at base of posterior triangle"rare"

5- Adson's test (not very accurate)

- May demonstrate compression of subclavian artery
- Ask the patient to extend his head, look at the opposite side & take a deep breath then palpates the radial pulse while the arm is pulled down.
- If the test is positive → the radial pulse strength is reduced.



Differential diagnosis

A) Other causes of localized pressure on the nerve

1. Cervical spondylosis.
2. Carpal tunnel syndrome.

B) Other causes of Raynaud's phenomenon

1. Raynaud's disease.
2. SLE and other collagen disease as they may be associated with vasculitis



Investigations

- 1- Plain X-ray to the neck → a bony cervical rib
- 2- Nerve conduction study between the neck and the forearm
 - Detect delay in conduction.
 - Differentiate between thoracic outlet & carpal tunnel syndromes

Treatment

Early cases (conservative)

Physiotherapy → strengthen shoulder muscles → relieve symptoms

Failure of conservative treatment

- **Surgery** → Aim: relieve the compression

- Methods:

- 1- Excision of a bony cervical rib.
- 2- Division of the scalenus anterior muscle (scalenotomy) to relieve the compression anteriorly.
- 3- Excision of the 1st rib to relieve the compression inferiorly.
(Very efficient & can be done through a trans-axillary approach)



Radiographic discovery of a cervical rib in asymptomatic person deserves no treatment

Radical block neck dissection

Definition

Removal of all LNs on one side of the neck in one mass (en bloc)

Aim

- Eradication of malignancy aiming at cure
- Achieved by removing the 1^{ry} lesion & its LN metastases

Indications

- 1- Operable 1^{ry} malignancy of head or neck with palpable malignant cervical LNs (**the main indication**)
- 2- Prophylactic radical block neck dissection
 - Cervical nodes are impalpable
 - Indications: malignancies that are known for their high tendency to lymphatic spread as carcinoma of the tongue and melanoma.
 - In other types of malignancies if LNs are not palpable → the 1^{ry} is removed then follow up the patient every 2 months, If LNs become enlarged → block neck dissection

The two most frequent neck swellings are:

I. Lymph node enlargement.

1. Acute lymphadenitis
 2. Chronic lymphadenitis (commonest is non-specific)
 3. Keep in mind more serious causes like TB, Hodgkin's disease and secondaries.
 4. Secondaries may arise from hidden malignancies:
 - a) Nasopharynx
 - b) Pyriform fossa
 - c) Nasal sinuses
 - d) Thyroid papillary carcinoma
- II- Thyroid enlargement (goiter)

A) Classic radical block neck dissection

- Done through different incisions, the popular is **goblet incision**

- Structures to be removed (removed in one mass "en bloc")

- 1- All lymph nodes on one side of the neck.
- 2- The sternomastoid muscle
- 3- The carotid sheath
- 4- The internal jugular vein
- 5- The submandibular salivary gland
- 6- The lower part of the parotid gland.

B) Modified radical block dissection

Similar to radical block neck dissection but with preservation of:

- 1- The internal jugular vein
- 2- Sternomastoid muscle
- 3- Accessory nerve

- Indications:

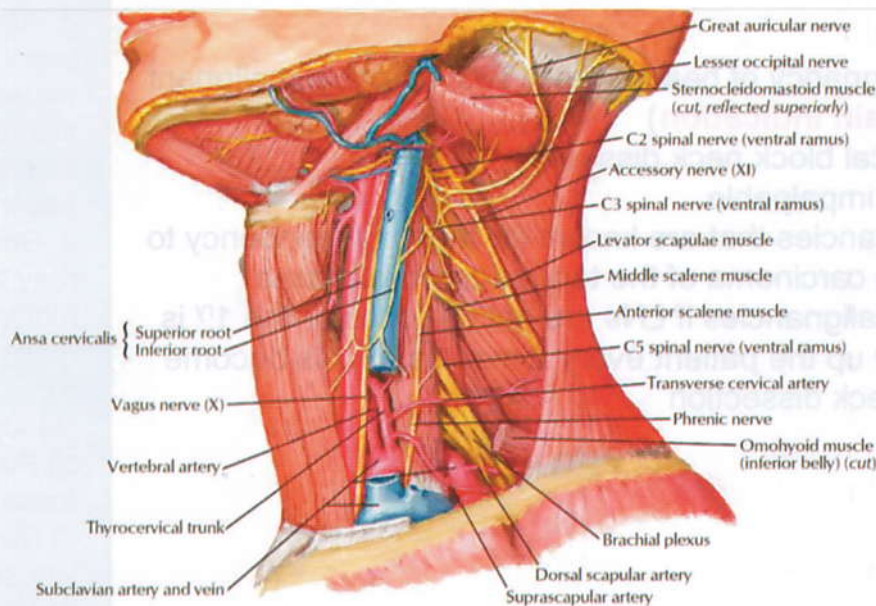
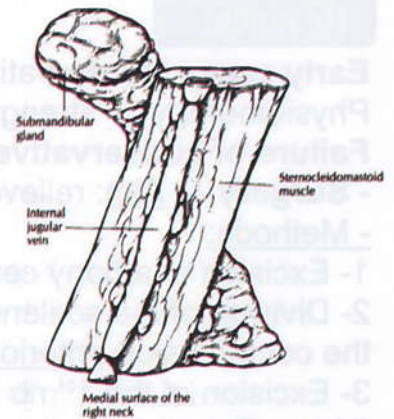
- 1- Thyroid cancer
- 2- Bilateral block neck dissection → Preservation of one jugular vein on the less affected side to provide adequate brain venous drainage

C) Suprahyoid block dissection

- Bilateral dissection to LNs above the level of hyoid bone
- The submandibular salivary glands are also removed

- Indications

- 1- Carcinoma of the lower lip.
- 2- In early cases of carcinoma of the tip of tongue & floor mouth



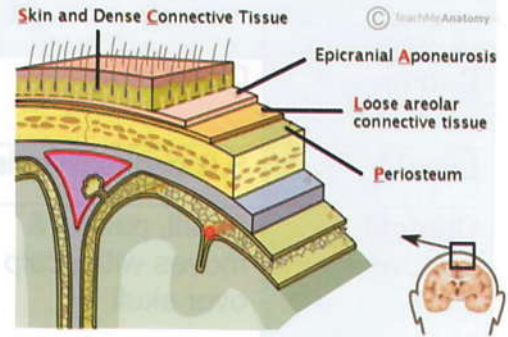
SCALP, SKULL AND BRAIN

Anatomy of the scalp

- The scalp is made of soft structures that cover skull
- Extension: from one temporal to the other and from the eyebrows to the superior nuchal line.

- Layers

1. **Skin.**
2. **Connective tissue** contains blood vessels & nerves of skin
3. **Aponeurosis** (**galea aponeurotica**) that contains occipito-frontalis muscle (2 pairs of bellies "frontal & occipital")
4. **Loose areolar tissue** contains few blood vessels
5. **Pericranium**: This is the periosteum on the outside of the skull



Scalp Injuries

- Wounds of the scalp include:
Contusions, haematomas, lacerations or incised wounds
- Characterized by:
1- Bleeding is very profuse up to shock
2- Caused by sharp or blunt instruments or falls on the head.
3- Healing is rapid.
4- If infection occurs in loose areolar tissue → extensive cellulitis
- Management:
1. If there is a possibility of a skull fracture → do plain skull X-ray
2. Closure is in 2 layers (galea to galea by absorbable sutures & Skin to skin by non absorbable sutures)
-If there is liability to infection, close in one layer of loose sutures
3- When there is scalp defect → do rotational flap

Infections of the scalp

- Etiology: 2^{ry} to wounds.
- An abscess may occur in S.C., subgaleal or subperiosteal layers
- Intracranial extension of infection may occur from the subgaleal area via the emissary veins.
- An infected sebaceous cyst is commonly seen in the scalp
- Treatment: 1- Antibiotics according to culture studies.
2- An abscess should be drained

Tumours of the scalp

1-Benign:

Lipoma-Papilloma-Plexiform Neurofibroma- haemangioma-Cirroid aneurysm

2- Locally malignant: Basal cell carcinoma

3- Malignant:

Epithelioma-Melanoma-Fibrosarcoma-Sebaceous adenocarcinoma-Metastases

Anatomical facts:

1. Scalp is rich in blood vessels → wounds heal nicely & without infection
2. Arteries present in the S.C. layer are adherent to the fibrous tissue of this layer → when a vessel is injured, the muscular coat of the divided artery can't retract → profuse bleeding
- 3- Skin, S.C tissue & epicranial aponeurosis are firmly attached to each other, but loosely connected to pericranium by loose areolar tissue → complete avulsion of scalp can occur.
4. Loose areolar tissue layer allows accumulation of large amounts of blood or inflammatory exudate

Haematoma of the scalp

	Subcutaneous	Subgaleal	Cephalhaematoma (subpericranial)
Cause	Direct blow	Direct blow	Head injury during delivery
Level	S.C connective tissue	Under galea	Under periosteum
Clinical picture	Small, painful & moves with scalp over skull	Large, soft fluctuating swelling reaching to the supraorbital ridges anteriorly, to the nuchal lines, posteriorly and laterally to the temporal lines (The scalp floats over the swelling)	Haematoma is limited by suture lines to the underlying bone usually parietal
Treatment	Resolve spontaneously	Aspiration and a pressure bandage	

How to stop bleeding from a scalp wound?

a. Direct pressure

- Exclude first depressed fracture

b. Applying multiple Allis forceps to the edge of the wound and bending them.

c. Applying multiple artery forceps to the galea aponeurotica and allowing them to fall back over the skin edge

d. All these measures are temporary before suturing of the wound.

Skull

Tumours of the skull:

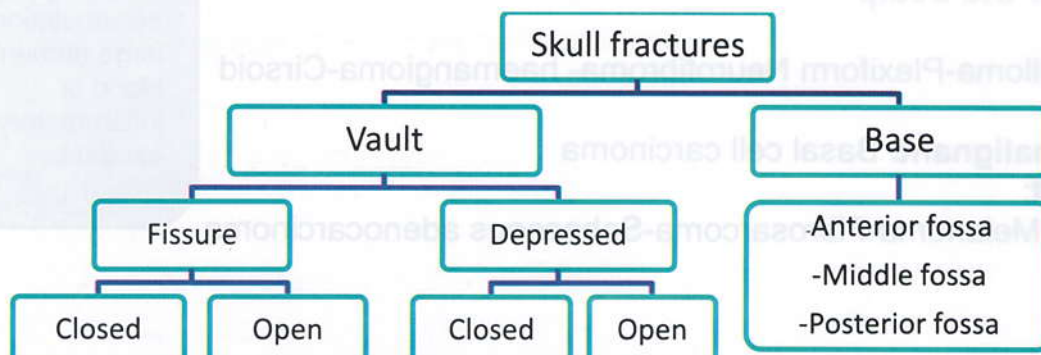
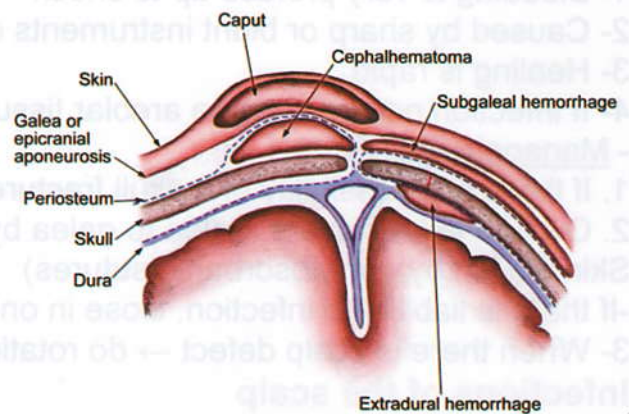
1- **Benign:** (rare) ivory osteoma occasionally arises in frontal sinus

2- **Malignant:** These are either:

A) primary :

Osteosarcoma, fibrosarcoma, giant cell tumour & multiple myeloma

B) Metastases: from the breast, thyroid, adrenal, kidney & prostate.



Fractures of the vault

Etiology

1. **Direct localized blow** over small skull area → closed or compound depressed fractures
2. **Compression by a hard flat surface** → fissure (linear) fracture
3. **Missile injuries**

Clinical picture

A) Fissure fracture: clinical picture of associated cerebral damage

B) Depressed fractures:

i) Closed: - Rare in adults

- Associated with overlying haematoma → may obscure the fracture
- The depressed segment rarely causes cerebral compression

ii) Compound:

- There may be profuse bleeding, leakage of CSF or brain prolapse
- Concussion is surprisingly slight and usually no compression.
- The main hazard here is the liability to infection

Investigation

Plain x-ray → reveal the fracture (D.D from suture line of the skull
→ exists at certain sites & has a serrated edge)

Treatment

A) Fissure fracture:

- Treatment of associated cerebral damage
- If present over the course of middle meningeal vessels → possibility of an extradural haematoma.
- If the fracture is compound, treat the scalp wound

B) Depressed fractures

I) Closed: - Essentially by conservative measures.

- Indications for surgery to raise the depressed fracture:

1. Large depressed segment > 1 inch with possibility of a dural tear.
2. Compression of an important area as motor or speech center.
3. Cosmetic deformity, e.g. in the frontal bone.
4. If the fracture is overlying an air sinus

II) Compound:

- Operated upon in theatre under absolute aseptic precautions:

1. Foreign bodies are removed.
2. Elevate the depressed segment gently to avoid dural tear
3. Suck any prolapsed or necrotic brain tissue & do haemostasis
4. Any dural tear is repaired.
5. Removed bone segments are cleansed and replaced.
6. The pericranium and the scalp are sutured
7. Prophylactic antibiotics are administered



Complications of a depressed fracture:

1. Dural tear → brain prolapses
2. Infection → may lead to osteomyelitis or meningitis.
3. Epilepsy (early or late)
Elevation of the depressed segment may Reduce but not prevent this complication
4. Cosmetic deformity.
5. Severe bleeding from any of venous sinuses

Fractures of the Base

Etiology

A) Indirect trauma (most common)

- Falls or blows on the vault → compression of skull at one area & giving way of skull in the opposite. As vault is elastic → it escapes while the base being rigid & weakened by multiple foramina, breaks. (Sometimes, fracture starts in vault & extends to base)

B) Direct trauma

- Due to penetrating sharp objects or bullets

Clinical picture

A) Anterior Fossa

1. Escape of intracranial contents

a) Blood:

i) Epistaxis if cribriform plate is fractured

ii) Lid ecchymosis (**Panda bear or Raccoon sign**) if orbit is fractured

- Distinguished from an ordinary black eye by :

1. There is no bruising of the skin around the orbit.
2. The effusion occurs several hours after the injury and commences in the lower lid before the upper.
3. Extravasation occurs into tissues at back of the orbit → pushes the eye forwards,
4. Extravasation may prevent ocular muscles action → limitation of eye ball movements
5. If extends in subconjunctival space → can't see its posterior limit (D.D from subconjunctival haemorrhage due to direct trauma)

b) CSF → **Rhinorrhoea** if dura is torn & cribriform plate is fractured (persistent salt taste due to the high chloride content)

- Air may enter cranial cavity when patient blows his nose (**pneumocephalus**)

c) Brain matter may escape into the nose or pharynx.

2- Cranial nerves injury

i) Olfactory nerve (if unilateral → unrecognized partial anosmia)

ii) The optic nerve usually escapes

iii) 5th nerves may be injured at the sphenoidal fissure.

iv) Third nerve palsy → a dilated pupil in a conscious patient.

3- **Associated brain injury** → Concussion (usually severe)

Essential clinical features

1. Escape of cranial contents, e.g. blood, CSF or brain matter
2. Injury to the cranial nerves.

All cranial nerves are liable to be injured **except the 12th** because the condyloid foramen is protected by a ridge of bone which deflects the line of fracture into the foramen magnum.

3. Associated brain injury depending on fossa involved

- They are often compound fractures & the risk of infection is high



B) Middle fossa

1. Escape of intracranial contents

a) Blood

- From the ear (the most common & characteristic sign) (D.D from the bleeding of ruptured drum)
- Epistaxis if fracture involves the nasal sinuses (commonly sphenoidal → cavernous sinus intimal carotid artery injury)
- Post auricular ecchymosis (**Battle's sign**)

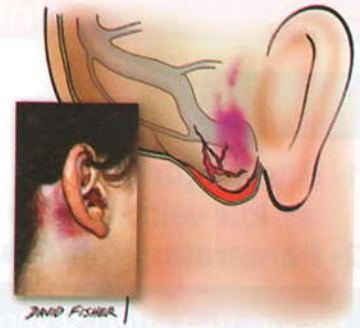
b) CSF → Otorrhea

c) Scalp emphysema around the ear if mastoid air cells affected

2- Cranial nerve injury

- The facial nerve (common)
- The 8th, 6th & 2nd and 3rd divisions of 5th nerve

3- Concussion with signs of severe intracranial damage



C) Posterior fossa

1. Extravasation of brain contents

- Blood in the suboccipital region → a **boggy swelling** or nape discolouration (External haemorrhage does not occur because the fracture is rarely compound)

2. Cranial nerves injury

- 9th, 10th & 11th nerves (hypoglossal nerve usually escapes)
- The upper cervical nerves are sometimes irritated by blood → head retraction and stiffness of the cervical muscles

3. Associated brain injury:

- Pons & medulla may be injured → deep unconsciousness
- Subtentorial haemorrhage → bulbar compression → death

Treatment

1. Prevention of infection

- Prophylactic antibiotics started at once & continued for one week after cessation of bleeding or CSF leakage.
- Avoid mouth or ear washes → increases the risk of infection.
- When the ear is involved → sponge daily with cotton wool.

2. Control of CSF leakage

- Semi-setting position in bed
- Usually stops in **85%** of rhinorrhoea & **95%** of otorrhea
- Surgical dural repair if discharge persists > 10 days

3. Treatment of the associated brain injury.

Blood from torn Tympanic membrane clots rapidly, but blood mixed with CSF continues to drip for several days without clotting

- Facial nerve injury can be either:

- Injured at time of the accident → permanent paralysis.
- Paralysis appears after a few days from compression by extradural Haemorrhage → often recovers
- Paralysis develops some weeks after injury due to pressure from fibrous tissue or callus → permanent

Intra-Cranial Injuries

Etiology

1- Blunt injuries:

- a) low velocity as in falls
- b) high velocity as in RTA

2- Penetrating injuries as in missiles.

Factors that affect severity of cerebral injuries

1. Brain distortion

- a) Posterior displacement → distortion hypothalamus & brain stem → affects reticular formation → loss of consciousness
- b) Anterior displacement → distortion of the corpus callosum → widespread damage to neurones, nerve fibres, glia & blood vessels

2. Mobility of the brain in relation to the skull and membranes as in Acceleration or deceleration injuries

3. Configuration of the interior of the skull.

- Smooth skull areas → less damage
- EX.: Temporal lobe may be damaged by the sharp sphenoid ridge and the frontal pole by the rough floor of the anterior cranial fossa.

4. Age of the patient

- A young patient → better chance of recovery

Pathology

A) Primary pathological sequelae

1. Cerebral concussion

- Slight brain distortion without any organic structural damage
- Immediately after the injury → temporary loss of consciousness

- Manifestations: 1- Muscles are relaxed

2- Pulse is rapid & weak

3- Respiration is shallow and slow.

4- Pupils are rounded, regular & reactive

5- Reflexes are absent or sluggish

- Lasts from few minutes to few hours → complete recovery

2. Cerebral contusion

- There are areas of bruising & swellings with localized or generalized edema & haemorrhage due to tearing of blood vessels

- The pia & arachnoid are **intact**

- **Clinically:** i) Prolonged period of unconsciousness & on recovery → headache, photophobia and confusion.

ii) Physical signs of focal neurological damage

3. Cerebral laceration

- The pia & arachnoid are **torn** with bloody effusion in the CSF

- The main factor which leads to most of the head injuries is cerebral tissues displacement & distortion at the moment of impact

- Factors that raise the intracranial pressure in patients with head injury:

1. Brain edema.
2. Intracranial haematoma (extradural, subdural or intracerebral)
3. Cerebral swelling due to local vasodilatation and loss of autoregulation
4. Rise of arterial PCO₂

- Cushing triad

1. Decrease of pulse
2. Decrease RR
3. Elevation of blood pressure

B) Secondary pathological sequelae

1. Brain edema

- Intracellular & extracellular fluid accumulation
- May be localized around the area of brain injury or it involve the whole brain → marked rise of intracranial pressure.
- **Clinically:** simulates brain compression due to a haematoma but without focal manifestations.

2. Intracranial haemorrhage

- Arterial or venous bleeding → intracranial haematoma (may be extradural, subdural or intracerebral)
- Severe trauma → many small vessels tearing → deep intracerebral haematoma with serious brain damage, edema & necrosis.

3. Vascular changes

- Intracranial pressure increases → disturbing in cerebral blood flow → ischaemic necrosis → brain edema

Cerebral infusion pressure = [mean B.P - intracranial pressure]

4. Herniation

a. Tentorial herniation: herniation of medial part of temporal lobe on the side of supratentorial mass → compression of:

- i) Oculomotor nerve (constriction then dilatation of the ipsilateral pupil) & midbrain.
- ii) Descending motor pathways from opposite hemisphere → hemiplegia on the same side of haematoma

b. Foramen magnum herniation:

- Herniation of infratentorial compartment → compression of vital centres in medulla → decrease of pulse & RR and elevation of blood pressure (Cushing triad) with severe headache & neck rigidity

5. Brain stem injury

- Either 1^{ry} damage or 2^{ry} to unrelieved supratentorial herniation
- **Clinically:** unconsciousness with extension spasms in all limbs, opisthotonos, tachycardia, small pupils, pyrexia & rapid shallow breathing
- With intensive treatment → recovery but with marked spasticity

6. Infection

- In patients with compound fracture or fractures of the base especially if there is CSF rhinorrhoea or otorrhoea
- **Complications:** i) Meningitis **2-3 days** after injury → fever & neck stiffness (D.D. from subarachnoid haemorrhage by lumbar puncture)
- ii) Brain abscess

7. CSF rhinorrhoea

- 2^{ry} to a fracture involving the sinuses associated with a dural tear
- Liable to be followed by meningitis.
- **Treatment:** i) Initially by antibiotics
- ii) Indications for surgical interference: a- Occurrence of meningitis
- b- Persistence of rhinorrhoea > 10 days
- c- Presence of a fracture involving frontal or ethmoid sinuses

Extracranial factors that affect cerebral injury:

1. Respiration

Respiratory inadequacy → hypoxia & increased PCO₂ → severe brain edema & venous congestion → irreversible changes.

2. Blood volume & blood pressure

Hypovolaemia → ↓ cardiac output → irreversible cerebral ischaemic damage

3. Fluids

Cerebral injury → blood brain barrier disturbances & inappropriate ADH secretion (IV infusion of hypotonic fluids here

→ lower plasma osmolarity → increase brain edema & swelling)

4. Temperature

Rise in body temperature → increase metabolic demands & metabolites accumulation → neurological state deterioration

Acute extradural haematoma

Etiology

- Trivial trauma to the side of the skull → fracture of the temporal or parietal bone.

- Source of bleeding: 1- Middle meningeal artery (50%)

2- Middle meningeal vein leading

Pathogenesis

Blood will pass in 3 directions:

1. Outwards → a boggy swelling under the temporalis muscle.

2. Upwards → over the parietal region

3. Downwards → into the middle fossa

Clinical picture

3 Stages

1. Stage of concussion

- A mild blow to the head → a brief period of concussion

2. Stage of lucid interval

- Patient recovers from the initial concussion & resumes his activities

- During which blood accumulates gradually in the extradural space

- If the source of bleeding is venous, the lucid interval will be longer.

3. Stage of compression

a. Gradual deterioration of consciousness → confused & irritable later drowsy, semicomatose or comatose with snoring

b. Compression of ipsilateral cortex → contralateral hemiparesis

c. Tentorial herniation → compression of oculomotor nerve → constriction followed by dilatation of the ipsilateral pupil. Later → contralateral pupil will constrict and then dilated

d. Continuous tentorial herniation → compresses crus against the rim of tentorium → hemiparesis on same side of the haematoma

e. As coma deepens → blood pressure rises while pulse & respiration slow down.

- Terminal stage → hyperpyrexia, decerebrate rigidity and bilateral dilated fixed pupils

An extradural haematoma can occur without a skull fracture in children as they have an elastic skull

- Acute Intracerebral Haematoma

- Incidence:

the least common

- C/P: usually accompanied by brain contusions, laceration, edema & necrosis → poor general condition

- Investigations:

CT scan

- Prognosis:

Poor

Acute Subdural Haematoma

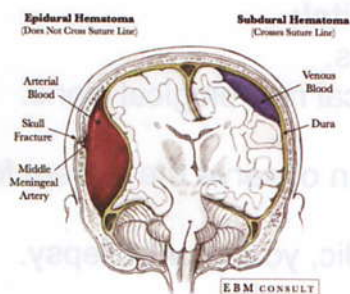
Etiology

1- Usually severe blow to the skull → rupture of a large vein as it crosses subdural space to reach the fixed subdural venous sinuses.

2- Cortical laceration (less commonly)

Clinical picture

	Acute extradural haematoma	Acute subdural haematoma
Trauma	Mild	Severe
Brain damage	Mild	Severe
Lucid interval	Usually present	Absent
CT scan		
a- Side	Usually unilateral	Common bilateral
b- Brain side	Convex	Concave
c- Density	Usually uniform	Less uniform
d- brain	Normal	Oedema
Prognosis	Good with early surgery	Bad (mortality >50%)



Management of intracranial injuries

1. Dealing with life saving priorities

- Protection of the airway (**most serious**)
- Maintenance of adequate breathing
- Arrest of haemorrhage
- Correction of shock (most likely due to internal haemorrhage in the thorax or abdomen)

2. Initial examination

(a) **Vital signs:** pulse, blood pressure and respiratory rate.

(b) **Scalp:** For any scalp wound or haematoma.

(c) **Skull:** for any fracture

(d) **Assessment of level of consciousness** by Glasgow coma scale

The higher the score, the better is the prognosis.

Classification according to the scale into:

I- Mild (13-15) ii- Moderate (9-12) III- Sever ≤ 8

Points	Eye response	Best verbal	Best motor
6	-	-	Obeys command
5	-	Oriented	Localizes pain
4	Spontaneous	Confused	Flexion withdrawal
3	To speech	Inappropriate words	Abnormal flexion (decorticate rigidity)
2	To pain	Incomprehensible sounds	Abnormal extension (decerebrate rigidity)
1	None	None	None

- The majority of patients with cerebral injury will need only conservative measures not surgical interference. (Unnecessary surgery → worsen the condition)

- Many extracranial factors (blood volume, P_{O_2} , PC_{O_2} & the temperature) have a serious impact on the injured brain.

(e) Pupils: examine both for their size and reaction to light.

- An immediately dilated pupil after the accident → direct orbit or the oculomotor nerve injury

- A constricted pupil on initial examination & later dilates → lateralization due to a supratentorial haematoma

(g) Limbs: Examine for any fracture or vascular injury.

- Hemiplegia in acute phase is likely to be due to 1st cerebral damage rather than due to a compressing intracranial haematoma.

(h) Chest: for fractured ribs, pneumothorax, or haemothorax

(i) Abdomen: Examine for internal haemorrhage or peritonitis.

(U) Back: For the possibility of fractures or dislocations.

3. Indications for admission to the hospital:

a. Any depression of level of consciousness.

b. Skull fracture

c. Focal neurological signs.

d. Persistent headache or vomiting.

e. Absence of responsible relatives who can observe the patient for the first 24 hours.

f. Difficulty in assessing the patient: alcoholic, young or epilepsy.

4. Investigations:

- Done after the patient is resuscitated and is stable

(a) Plain skull x-ray → site & type of a skull fracture.

- A foreign body can be visualized

(b) CT scan is highly recommended for:

i- Depressed or compound fractures.

ii- Impaired level of consciousness or focal neurological signs.

iii- Basal skull fractures.

iv- Deteriorating level of consciousness.

v- Vomiting more than once.

vi- Seizures.

- CT scan will detect brain edema, brain contusion or laceration.

- Assess the site, size & progress of an intracranial haematoma

- Acute extradural or subdural hematomas are hyperdense

compared to the brain & there may be shift of mid line structures

5. Continuing care and observations:

-The aim of conservative treatment:

i- Give the patient the maximum care until spontaneous recovery

ii-Detect complications as early as possible

(a) Attention to the airway & if spontaneous respiration is inadequate to keep normal levels of PO_2 and PCO_2 → insert an endotracheal tube which needs full sedation or muscle relaxants .

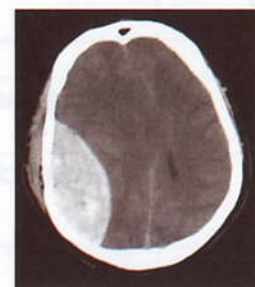
- It can be left up to 7 days. If ventilation is needed for a longer period → do a tracheostomy

Any patient with GCS ≤ 8 needs intubation.

The most important sign of patient's progress → level of consciousness

- CT scan should be done before surgery to evaluate the size of the haematoma & degree of brain damage and edema

- If the patient is in urgent need for evacuation of an intracranial haematoma, no time should be lost in doing investigations, and surgery should be done immediately.



(b) **A Foley catheter** is inserted to estimate the urine output.

(c) **Frequent change of posture** to avoid bed sores.

(d) **Physiotherapy** to the joints and massage to the muscles.

(e) **A nasogastric tube** is inserted for feeding.

(f) **Osmotic diuretics:**

- Aim: raise the plasma osmolarity → reduce brain edema

- Method: 250 ml of 20% Mannitol over 20 minutes and may be repeated every 8 hours (should not be used for > 48 hours to prevent rebound edema & electrolyte disturbances)

- Before giving mannitol → exclude intracranial haematoma and check that the renal function is satisfactory.

(g) **Frusemide:** 40-80 mg IM is an alternative to mannitol.

6. Repeated observations for of the following:

i- Level of consciousness using the Glasgow coma scale.

ii- Pulse, B.P and temperature

iii- Respiration

iv- Pupils

v- Reflexes

Causes of deterioration of the patient:

1. Brain edema → increased intracranial tension

2. Airway obstruction and/or hypoventilation → brain swelling and increased intracranial tension.

- Respiratory insufficiency confirmed by estimating PO_2 & PCO_2

3. Intracranial haematoma → confirmed by CT scan.

4. Fever due to respiratory infection or meningitis.

5. over transfusion by hypotonic fluids or dehydration.

6. Epilepsy

7. Surgery to evacuate an acute intracranial haematoma:

Early evacuation before deterioration and fatal herniation of the cerebellar tonsils and medulla through the foramen magnum occurs

- A rising intracranial tension produces the following manifestations:

1. Deterioration in the level of consciousness.

2. Slowing pulse rate.

3. Rising blood pressure.

4. Slowing respiration.

Operation:

1. Under general anaesthesia with endotracheal intubation.

2. The patient is placed supine with the site of haematoma uppermost & head is raised slightly to reduce venous bleeding

3. A formal craniotomy by an osteoplastic flap is done

-The flap includes skin, temporalis muscle and part of skull.

- 4 or 5 burr holes are done and are connected by a saw.

4. Skin and temporalis are not dissected from bone.

- The osteoplastic flap is turned down → exposing dura.

5. If an extradural haematoma is present → removed by suction and the middle meningeal artery coagulated or underrun by a stitch.

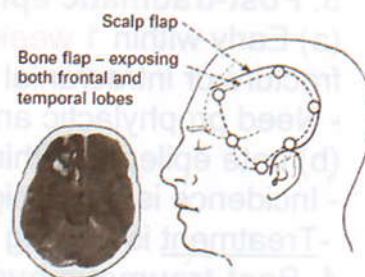
6. Occasionally it may be necessary to follow the middle meningeal artery to foramen spinosum which is packed with bone wax

Corticosteroids are empirically prescribed but there is no evidence that they reduce the cerebral edema nor improve the outcome in severe head injury

- If epilepsy is not accompanied by convulsions, it is difficult to D.D. it from an intracranial haematoma

- A high intracranial pressure is caused either by haematoma or cerebral edema

- CT scanning can easily diagnose the presence & site of intracranial haematoma



7. If a subdural haematoma is found → open the dura mater in a cruciate fashion → rapid decompression of the brain
8. A drain is placed and the flap is returned to its place and fixed by suturing temporalis fascia and skin.
9. Prophylactic anticonvulsant drugs are prescribed for 6 weeks

Late Complications of Head Injuries

1. Chronic subdural haematoma

- Incidence: in elderly & alcoholics 2^{ry} to a slight blow to the head which may pass unnoticed
- Bilateral in **50%** of cases
- Pathogenesis: sudden brain displacement → rupture of superior cerebral veins as they pass to the venous sinuses → collection of blood in the subdural space.

Clinical picture

Symptoms (appears **weeks to months** after trauma)

- Vague and consist of: chronic headache, mental apathy, slowing of cerebation and even stupor

Signs 1- May be absent

- 2- May be unilateral or bilateral extensor planter response
- 3- Pupillary changes (late and denote impending conization)
- May be diagnosed as psychosis or cerebrovascular accident

Investigations

- 1- CT scan → haematoma which is hypodense compared to brain
- 2- Fundus examination → Papilloedema is exceptional

Treatment

By evacuation through burr holes

2. Post-traumatic headache

- With serious head Injury → some residual as headache, giddiness, impaired sleep and defective concentration.
- These patients need prolonged convalescence.

3. Post-traumatic epilepsy: may be either →

(a) Early within **1 week** after the injury (especially depressed fracture or intracranial haematoma)

- Need prophylactic anticonvulsants for **6 weeks**

(b) Late epilepsy within **1-4 years** after the injury

- Incidence is much higher in those who had early epilepsy.

- Treatment is by long term anticonvulsant drugs.

4. Post-traumatic hydrocephalus → may need shunt operation

Cerebral atrophy in elderly persons and alcoholics makes brain displacement easier



Hydrocephalus

Definition (It literally means 'water head')

Abnormal accumulation of excess cerebrospinal fluid (CSF) within the ventricles and/or the subarachnoid space

Hydrocephalus in Children

Prevalence

- As an isolated congenital disorder in approximately **1/100** live births
- In association with spina bifida in **1/1000** births.

Pathophysiology

Excessive CSF accumulation → dilatation of the ventricles and increased intracranial pressure

- Usually results from an obstruction in the CSF pathway and rarely from overproduction of CSF

Classification

1. Non-communicating hydrocephalus: due to lesions that obstruct the ventricular system either at the aqueduct or basal foramina
2. Communicating hydrocephalus: due to lesions that obstruct the subarachnoid space

Etiology

1. Aqueduct occlusion due to aqueductal forking (a congenital anomaly) or subependymal gliosis after toxoplasmosis
2. Arnold Chiari malformation: The 4th ventricle is displaced caudally below the foramen magnum → impedance to CSF flow to the subarachnoid space (commonly associated with myelomeningocele)
3. Dandy-Walker syndrome: cystic dilatation of 4th ventricle → obstructs CSF flow
4. Cerebral malformations causing arachnoid cysts as encephalocele or hydrancephaly → obstruct the CSF flow
5. Neoplasms mainly in the 4th ventricle (medulloblastomas, astrocytoma and ependymomas).

Clinical picture

- 1- If occurs before closure of skull sutures → the infant presents with macrocephaly, tense nonpulsatile anterior fontanelle, distended scalp veins & separated cranial sutures.
- The eyes show the **sun set** appearance with failure of upward gaze
- 2- If sutures have closed → presents with headache awaking him from sleep, vomiting, drowsiness, blurred vision & squint

Normal CSF circulation

- Production:

by the choroid plexus in the lateral ventricles of the brain.

- Pass way:

through the foramen of Monro on each side to reach the midline third ventricle → aqueduct of Sylvius → 4th Ventricle → subarachnoid space around Brain & spinal cord (4th ventricle has two lateral foramina of **Luschka** & one mid line foramen of **Magendi**)

- Absorption: inside the skull through the arachnoid villi to the blood stream.



Investigations

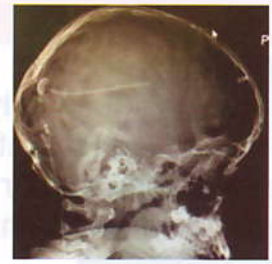
1. X-ray of the skull → separation of sutures in infants and the "beaten silver" appearance in older children.

2. CT and MRI (of choice)

- Diagnose, detect site of obstruction and causative pathology

3. Cranial ultrasonography (through the anterior fontanelle)

Useful for follow up



Treatment

- Removal of the cause but this is impossible in most cases of congenital hydrocephalus → so divert CSF using shunts:

a) Ventriculo-peritoneal shunt (most common)

- Consists of: a ventricular catheter, a valve permitting CSF flow in one direction and a distal peritoneal tube.

- The valve opens when the intracranial pressure exceeds a certain level → excess fluid enters the peritoneum → absorbed there to reach the systemic circulation.

- The catheter is inserted through a burr hole in the skull to reach the frontal horn of the lateral ventricle.

- The peritoneal tube is inserted in the epigastric midline below the xiphoid process

b) Ventriculo-atrial shunt

c) Ventriculo-pleural shunt.

- The commonest complications of shunts:

1- Infection

- Treatment: i- remove the shunt, an external ventricular drain is applied and give IV antibiotics for **2 weeks**.

ii- After infection subsides → insert another shunt on the other side of the head.

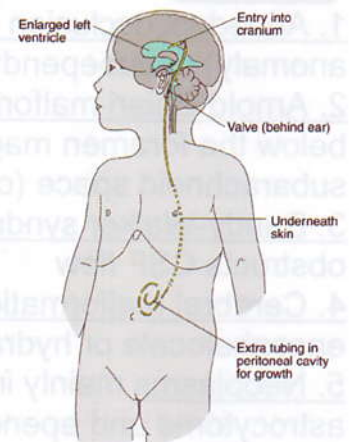
2- Obstruction

- Clinically: the child develops increased intracranial pressure.

- Treatment: revising the shunt & replacing the malfunctioning component

Hydrocephalus in Adults

Ventriculoperitoneal Shunt Placement



Classification and etiology

(a) Non-communicating:

- Tumours or cysts of ventricles
- Aqueduct stenosis
- Posterior fossa malformations & tumours

(b) Communicating

- Trauma
- Subarachnoid haemorrhage
- Infection
- Idiopathic
- Extra-axial tumours

Clinical picture

- 1- In acute hydrocephalus → headache, vomiting, papilloedema, & drowsiness
- 2- In chronic & normal pressure hydrocephalus → gait disorders, memory loss, urinary incontinence & slowing of thought and action

Investigations

- 1- CT and MRI (**investigations of choice**)

Diagnose hydrocephalus and its underlying cause.

- 2- An overnight monitoring of CSF pressure and perfusion

-In case of normal pressure hydrocephalus → record bouts of raised intracranial tension (Access is gained through a ventricular or a lumbar catheter)

Treatment

CSF diversion is done by:

- 1- Ventriculo-peritoneal shunt (mainly)
- 2- Ventriculo-atrial or Ventriculo-pleural shunts (less commonly)
- 3- Lumbo-peritoneal shunt (for the communicating variety)

Brain Abscess

Etiology and pathogenesis

Routes of infection:

1. **Direct spread** (**the majority**) from paranasal sinuses, middle ear or mastoid infection
2. **Skull defects** which are acquired (e.g. cholesteatoma of middle ear) or congenital (e.g., dehiscence of the tegmen tympani).
3. **Haematogenous spread**
-Source: 1st site from dental or tonsillar abscess → usually causes multiple brain abscesses.
- Commoner in congenital heart disease with right to left shunt
4. **Post-traumatic** especially if foreign bodies are present
5. **Previous craniectomy:** especially in immunocompromised patients taking immunosuppressive therapy for transplantation

Clinical picture

General: low grade fever, severe headache resistant to analgesics

Neurological:

General: symptoms of increased intracranial tension, alteration of the conscious level in about **50%**

Local: focal neurological symptoms

The formation of a collagen capsule in a developing abscess is the single important response that limits the spread of infection to rest of the brain.

Investigations

A) Laboratory findings

Elevated WBCs and sedimentation rate

B) Radiological

CT or MRI (investigation of choice)

- CT scan is done with and without contrast.
- The MRI is done with gadolinium enhancement.
 - They show a single (or multiple) space occupying lesion that is well delineated with an enhancing wall.
- The differential diagnosis of:
 - 1- Single brain abscess → solitary metastasis or cerebral infarction
 - 2- Multiple abscesses → multiple metastases and tuberculomata

Treatment

A) Non-surgical treatment:

- Indication: an abscess that is < 1.5 cm.

- Method:

1. **Antibiotics:** high dose of broad spectrum antibiotics until results of culture and sensitivity appear
 - The chosen drugs should be able to cross the blood brain barrier
 - The usual regimen is a combination of penicillin G, trimethoprim sulphamethoxazole and an aminoglycoside.
2. **Corticosteroids** → reduce cerebral edema despite their potential adverse effect on the infection

B) Surgical treatment

Excision versus Aspiration (done by using a blunt-tipped needle by way of a burr hole in the skull)

• Factors that favour aspiration:

- 1- Multiple abscesses.
- 2- A deeply seated abscess.
- 3- A critical location (e.g., motor or speech area).
- 4- Poor general condition of the patient.

• Factors that favour excision

- 1- Multilocular abscess (difficult to aspirate all the locules)
- 2- A superficial abscess.
- 3- The presence of a foreign body
- 4- Fungal abscesses (have poor response to antifungal therapy)

Lumbar puncture is contraindicated in cases of brain abscess to avoid a fatal conization

- Majority of responsible organisms are obligate anaerobes with **50%** of them resistant to penicillin.
- The mortality rate resulting from aspiration alone, aspiration followed by excision and 1st excision alone are almost similar

Intracranial Tumours

Clinical picture

1. May be symptomless.
2. General impairment of the cerebral functions such as amnesia, irritability, confusion or dementia.
3. Headaches (in about **1/3** of tumour patients)
 - Due to increased intracranial pressure or direct stretch of meninges
4. Evidence of increased intracranial pressure as:
 - i) headache
 - ii) Vomiting (not preceded by nausea, projectile, not related to meals, and occurs in the early morning)
 - iii) Blurring of vision ($\uparrow$ pressure $\rightarrow$ papilloedema $\rightarrow$ optic atrophy)
5. Specific syndromes are related to the site and nature of tumour:
 - a. Hemiplegia in tumours of the motor area.
 - b. Aphasia in tumours of the speech area.
 - c. Hormonal disturbances in functioning pituitary tumours (Cushing's disease, Acromegaly & galactorrhoea with prolactinomas)

Classification

2nd brain tumors $\rightarrow$ commonly from bronchial & breast cancers

Primary brain tumors

Glial tissue	Gliomas Astrocytoma Oligodendroglioma Epindymoma Glioblastoma multiforme
Meninges	Meningioma
Nerves	Neuromas, e.g., acoustic neuroma
Blood vessels	Haemangioblastomas
Embryonic	Medulloblastoma
Anterior pituitary	Pituitary adenoma Non-functioning adenoma Functioning adenoma
Maldevelopment	Craniopharyngioma (from Rathk's pouch)

Investigations

1. **Plain skull x-ray:** Either normal or show changes due to increased intracranial pressure in the form of:
 - a. Prominent convolutional markings.
 - b. Thinning and erosion of the dorsum sellae.
 - c. Widening of the cranial sutures (in infants before their closure).
 - Local changes may occur with specific tumours, e.g.:
 - i) Supracellar calcification in craniopharyngiomas.
 - ii) Hyperostosis and skull thickening in meningiomas
 - iii) Widening of skull base foramina with skull base tumours.

General considerations

The term intracranial tumour refers to all neoplasms arising from skull, meninges, blood vessels, pituitary and pineal glands, cranial nerves, brain tissue or congenital rests & metastatic tumors

Epidemiology:

- Commonest intracranial neoplasm is **metastasis**
- In $>20\%$ of all patients with cancer, the brain and its coverings are involved by metastases at sometime in the course of the illness
- In children, brain tumours are the 2nd in frequency to leukaemia representing **22%** of all childhood cancers

2. Computed tomography (CT).

-IV iodinated contrast is required in **60 to 80%** of examinations:

- I - CT accurately detects the site of the tumour.
- II - It clearly demonstrates its boundaries.
- III - It shows whether there is surrounding edema or not.
- IV - It demonstrates any shift of midline structures by the mass.
- V - It shows any associated hydrocephalus.
- VI - It shows any bony changes.
- VIII - It gives idea about tumour nature (solid, necrotic or calcified), and consequently its pathology

3. Magnetic resonance imaging (MRI)

- Contraindicated in the presence of metal implants as cardiac pacemakers, aneurysm clips, metallic FBs and patient support systems as respirators or intravenous line stands → material may move or stop working → endangering the patient's life
- Materials made of titanium permit safe MRI examination

4. CT angiography

Management

1. If the patient shows signs of compression → urgent dehydration using diuretics as 25% dextrose or mannitol.
 - Dexamethasone is very useful in treatment of edema surrounding brain tumours → restore a comatosed patient to consciousness while preparing for surgery.
2. If an intracranial tumour is associated with hydrocephalus (common with posterior fossa tumors) → Ventriculo-peritoneal shunt before or after surgery (depending on patient's general condition & surgeon's experience)
3. Many of the skull base tumors that were considered difficult to reach are now easily accessible by removing as much as possible of the bones of the skull base, face & ear to minimize brain retraction
4. CT or MRI guided stereotactic surgery is done for:
 - a) biopsy
 - b) Excision if the tumor deeply seated in the brain.
 - A head frame is used to facilitate accurate three dimensional localization of the lesion which can then be reached by fine instruments introduced through a burr hole.
5. In cases of malignant brain tumours or metastases → adjunctive therapy → improve the prognosis.
 - Radio and/or chemotherapy are supplementary to surgical excision

The most significant findings by examination pointing to an Intracranial tumours are:

1. Presence of papilloedema
2. Signs of focal damage to nervous system

- Lumbar puncture is contraindicated in suspected cerebral tumour → may precipitate a fatal conization of brain stem through the foramen magnum.

Individual intracranial tumours

Metastatic tumours

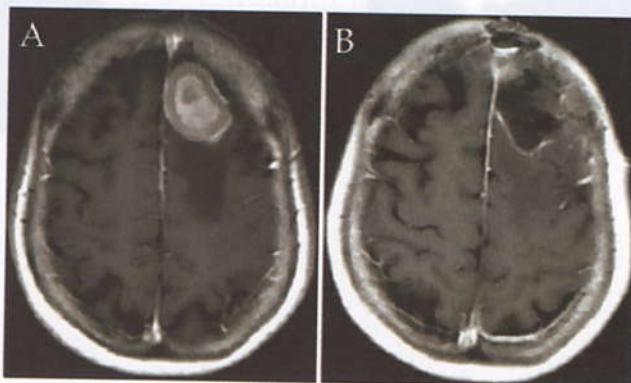
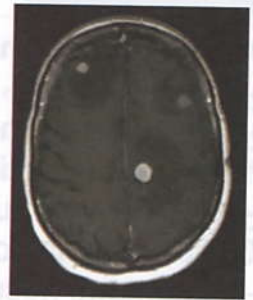
1. Multiple brain metastases → treated by brain irradiation.
2. Solitary brain metastasis:
 - a. **Disseminated** disease or life expectancy < 2 months → Brain radiotherapy
 - b. **Limited** systemic disease and life expectancy > 2 months
 - i. If surgically accessible → excision followed by radiotherapy
 - ii. If the lesion is inaccessible → only radiotherapy

Gliomas

- Definition: central nervous system tumors
- Cell of origin: cells of glial origin.
- Incidence: the commonest of primary CNS neoplasms
- Include: astrocytic tumours, Oligodendroglioma, ependymomas and glioblastoma multiforme
- Diagnosis: A biopsy or surgical resection
- Treatment: Adjuvant radio or chemotherapy is usually needed after biopsy or surgical resection (**Except for low grade astrocytomas**)

Meningiomas

- Incidence: a) **15%** of intracranial tumours
- b) Occurs in 4th to 6th decades c) Slight female preponderance
- Origin: from the arachnoid villi.
- Microscopically: appear as whorls of fibroblasts around a central hyaline material → calcify and form psammoma bodies
- Malignancy defined by frequency of mitosis, invasion of brain & metastases.
- The common sites: parasagittal sphenoid wing & olfactory groove regions.
- The tumour tends to produce hyperostosis of the skull.
- Course: usually long & generally benign
- Clinical picture: related to those of an intracranial mass or seizures
- Investigation: CT scan (accurate)
- Treatment: Complete surgical removal → cure



Pituitary tumours

Classification

A) Non-functioning pituitary adenomas

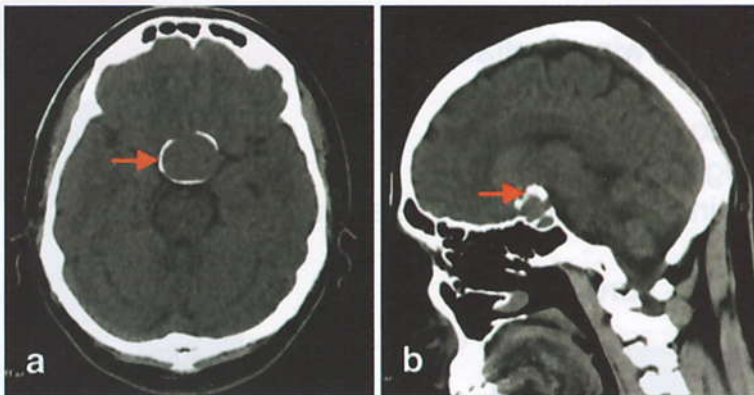
- Incidence: 30% of pituitary tumours & seen in 4th – 5th decades
- Other names: null cell tumours, undifferentiated tumors & none hormone producing adenomas.
- Presentation: (usually late until they are very large)
Optic chiasma compression → visual field deficits
- Treatment: microscopic trans-sphenoidal excision

B) Functioning pituitary adenomas

- Classification according to the hormones they secrete:
 - 1- Prolactin-secreting adenomas.
 - 2- Growth hormone-secreting adenomas → Acromegaly or gigantism
 - 3- Glycoprotein-secreting adenomas → excess TSH, LH or FSH.
 - 4- ACTH-secreting adenomas → Cushing's disease.
 - 5- Some of these secrete more than one hormone as prolactin growth hormone-secreting adenomas.
- Classification according to the size:
 - 1- Microadenomas < 1 cm
 - 2- Macroadenomas that have a larger size.
- Diagnosis:
 - 1- Clinical changes
 - 2- Hormone assessment
 - 3- Radiology → MRI & CT scan
- Treatment: excision by trans-sphenoidal root (whether sublabial or transnasal)
- In invasive tumour with incomplete excision → radiotherapy

Prolactinoma

- Incidence:
40% of all pituitary adenomas.
- C/P:
 - i) **In women**: amenorrhoea, galactorrhoea & infertility
 - ii) **In men**: impotence or may be asymptomatic.
- Hyperprolactinaemia in presence of macroadenoma does not necessarily mean that the tumor is prolactinoma
- Mild prolactin elevation < 150 ng/mL can be due to pituitary stalk compression
- Treatment:
Bromocriptine (anti-prolactin drug) → replaced surgery except for specific situations



SURGERY OF NERVES

Nerve Injuries

Etiology

A. Open injuries: knives, scissors, missiles and burns

B. Closed injuries:

a. Contusion by sudden direct violence.

b. Compression against adjacent bone by tourniquets or splints

c. Traction (stretching) such as tearing of the lateral popliteal nerve in forcible adduction of the knee or tearing of roots of brachial plexus in birth injuries

d. Fractures & dislocations → contusion, compression, stretch or laceration by the displaced bone.

e. Ischaemia from damage or occlusion of the main artery.

f. Accidental injection of irritant substances as in the case of the sciatic and radial nerves

Pathology

1- Neurapraxia (Functional block of nerves)

- Definition: functional paralysis of conduction but the nerve is anatomically intact without any organic rupture.

- Etiology: a. minor traction or compression injuries

b. Concussion & vibratory effect of a high velocity missile

- Pathology: fibres are intact with no axons degeneration

- Clinically: a. complete motor paralysis along nerve distribution

b. Patchy sensory loss or even no sensory disturbance at all

c. Electrical excitability of the paralysed muscles remains normal.

- Recovery: spontaneously within days or weeks

2- Axonotmesis

- Definition: partial or complete rupture of nerve fibres within an intact sheath

- Etiology: contusion & traction injuries or fractures and dislocations

- Pathology:

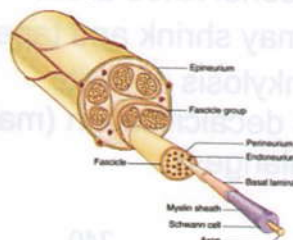
a) Damage of nerve fibres and Wallerian degeneration in distal portions of torn axons → complete motor & sensory paralysis

B) Regeneration of axons across the site of damage and continues distally to nerve endings at a rate of approximately 1 mm per day

with two delays: i- initial delay of about 10 days
ii- Final delay of 3 weeks when axons reach the synapse

- Recovery: usually complete because the axons remain in their respective neurolemmal tubes

- The length of time required for the recovery depends upon the level of the lesion



Anatomy

- A nerve trunk is formed of nerve fibres arranged in bundles bound by connective tissue fascia called

epineurium

- Each bundle is surrounded by connective tissue called perineurium

- Connective tissue around individual nerve fibres is called

endoneurium

- Each nerve fibre consists of the central axis cylinder surrounded by myelin and neurolemmal sheaths.

- Characters of peripheral nerve fibres:

1. May be motor, sensory, vasomotor or Sudomotor
2. Divided tracts can regenerate & recover to variable extent depending on trauma severity

3- Neurotmesis

- Definition: partial or complete anatomical division of the nerve.

- Pathology: Degenerative changes occur in the nerve as in axonotmesis but the gap fills with a mass of fibrous tissue and regenerating axons known as bulb neuroma.

- Complete division: i- terminal neuroma on the end of proximal segment separated by an interval from the atrophic distal end.

ii- Wallerian degeneration in the distal segment occurs.

iii- Retrograde nerve degeneration until 1st node of ranvier

- Complications: fibers mix in attempts of recovery

- Recovery: (Spontaneous recovery is impossible)

i- **Poorest** in mixed nerves supplying large number of muscles concerned with fine movement as ulnar & median nerves and in motor nerves which supply a large number of small muscles (due to maldistribution of fibres)

ii- **Best** in purely motor nerves which supply few groups of large muscles such as the radial

Clinical picture

A) Motor effects

1- Deformity (as claw hand deformity of ulnar paralysis)

2- Loss of voluntary contraction of supplied muscles.

3- Muscle atrophy

4- Loss of reflexes (superficial or deep)

B) Sensory effects

1- Anaesthesia: due to the overlap of dermatomes → area of anaesthesia is less than nerve anatomical distribution

2- Pain: (a favourable sign)

i- referred to area of cutaneous distribution of injured nerve

ii- More common in partial than in complete lesions

C) Autonomic effect

1- Causalgia

2- Sudomotor effects: anhidrosis in the denervated part over a larger area than the area of anaesthesia.

3- Vasomotor effects: Vasodilatation → skin becomes red and warm
Followed by vasoconstriction after about 3 weeks → blue & cold skin

D) Trophic changes:

- Etiology: disuse, sensory loss & vascular changes

- Effect: 1- skin loses its wrinkles → smooth, thin & inelastic

2- Normal resistance to trauma is lost and ulceration occurs

3- The nails become distorted and brittle.

4- Loss of hair in the denervated areas 5- loss of S.C fat

6- Tips of the fingers may shrink and taper.

8- Contracture and ankylosis of joints

9- Progressive bones decalcification (marked in proximal & distal extremities of the phalanges)

Investigations

1. Nerve conduction velocity (NCV)

I- Normal NCV is **50-70 m/sec**.

II- Neurapraxia does not interfere with NCV.

III- Decreases after neurotmesis (**20-40%** of normal after 1 month)

IV- Gradually increase during regenerating

2. Electromyography

I- Normally at rest → no electrical activity

II- During full voluntarily contraction → 500 milli volt recorded

III- **2-4 weeks** after complete nerve injury → spontaneous fibrillations

IV- In complete fibrosis → no electrical activity

3- Tinnel's sign

- Tapping the nerve → tingling sensation at the site of regeneration

Treatment

A) Conservative treatment

- Indications: neurapraxia & axonotmesis

- Aim: 1- prevent muscle stretching

2- Minimize muscle wasting

3- Preserve joint mobility

- Measures:

1. Splintage → prevent overstretching of the paralysed (removed at least once a day to permit all the joints to be put through a full range of passive movement)

2. Physiotherapy → prevent edema & joint stiffness

3. Electrotherapy → minimize muscle wasting and fibrosis

4. Skin protection (anaesthetic skin susceptible to be injured)

B) Surgical Treatment

- Indications: 1. Open nerve injuries.

2. Closed nerve injuries if:

a. Failure of recovery at a rate of 1 mm/day.

b. Failure of progress of Tinnel's sign.

c. If there is a palpable neuroma

- Timing of surgery:

1. Primary nerve suture

Indication: clean incised wounds

2. Secondary nerve suture

Indication: contaminated or lacerated

Timing: after complete skin healing (because nerve suture needs mobilization of both ends & opening new tissues planes → allow spread of infection)

- Primary operation is done to approximate divided ends & close the skin → early healing, preserve fibres orientation & prevent retraction

- The idea of NCV is to measure time that elapses after nerve stimulation at a chosen point to induce muscle action potential
- Electro-myography is done by inserting a needle in a muscle supplied by the nerve to be studied

- Prognosis of nerve repair in upper limb:

1. **Best** →

Radial

2. Intermediate
→ median

3. **Worst** →
ulnar

Technique:

1. General anaesthesia with a tourniquet.
2. A long incision.
3. Mobilization of the nerve ends.
4. Resection of the neuroma & damaged parts of nerve ends using a sharp knife.
5. Nerve suture. Either by:
 - a. **Epineurial repair** → Interrupted 6/0 prolene sutures pick up the nerve sheath only (epineurium).
 - b. **Interfascicular repair** → passing the sutures through the perineurium using the operating microscope

6. Nerve grafting

Indication: wide gap that cannot be approximated

Nerves used: small cutaneous nerves usually sural or ilioinguinal

Method: nerve is divided into segments that match the size of the defect → multiple nerve grafts (2-4) bridging the defect looking like a cable → called "**cable grafting**" & need vascularized bed for taking

Results depend on:

1. The injured nerve

Radial nerve is the best because it is predominantly motor & there is less chance of maldistribution of the fibres during regeneration

2. Level of suture: High-level injury → long recovery duration

3. Time interval between injury & suture.

The earlier the repair, the better is the results

4. Extent of injury

Large gaps between cut ends have a bad prognosis.

5. Age & patient's general condition: good in children & adolescents

Secondary Treatment

Operations to improve function when nerve recovery is impossible

1. Nerve transfer

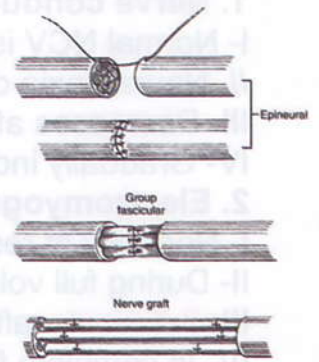
- Taking a nerve or branches of a nerve of less important role and transferring them to restore function of a damaged crucial nerve

2. Tendon transplantation, e.g., for radial palsy

3. Functioning free flap.

4. Arthrodesis e.g. arthrodesis of the shoulder for paralysis of the abductors and arthrodesis of the tarsal joints for paralytic talipes.

5. Amputation, e.g., amputation of the arm for total brachial plexus injuries and amputation of the leg for sciatic lesions



Late suturing of injured nerve → long period of muscle denervation → muscle wasting and degeneration → muscle deprived of nerve supply for > 2 years → cannot recover due to irreversible changes in motor end plates

Facial nerve injury

Etiology

A) Intracranial lesions may be:

- 1- **Supranuclear** → UMNL of lower half of the opposite side of the face only (since occipitofrontalis & orbicularis oculi muscles are bilaterally innervated)
- 2- **Nuclear** → the whole face & 6th nerve on the same side are affected → atrophy of facial muscles with crossed hemiplegia, (since the motor decussation takes place at a lower level)
- 3- **Infranuclear** → paralysis of same side of face together with deafness and cerebellar signs (results from pressure of a tumour in Cerebello-pontine angle on the root of the nerve)

B) Cranial lesions

- Fractures of base of skull, middle ear disease or operations on mastoid antrum → damage of intraosseous portion of the nerve
- In this part, any lesion which affects the nerve → also involves the auditory nerve, the petrosal nerves or the chorda tympani.

C) Extracranial lesions:

I-Commonly involved by Bell's palsy probably due to herpetic neuritis and may follow exposure to cold or air draught.

- **Pathogenesis:** Swelling within the nerve sheath → extends into stylomastoid foramen → nerve compression within its bony canal
- **Prognosis:** 1. Absorption of the exudate usually occurs before the pressure has damaged the nerve permanently
- 2. In about **10%** of cases → some degree of paralysis persists.

II- The nerve trunk or branches damage by: injuries, operations (Parotidectomy) or malignant tumours in the parotid region

The nucleus of facial nerve is in the floor of the 4th ventricle.

Clinical picture

1- Face: The affected side is flat, motionless & expressionless:

- a) Loss of forehead corrugations on looking upwards the
- b) No contraction on laughing or showing teeth → asymmetry

2- Eye

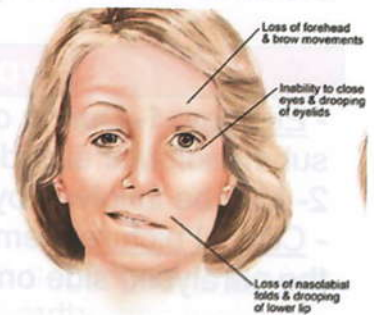
- Cannot be closed & on attempting to do so → eyeball rolls upwards and outwards → corneal ulceration due to exposure
- Epiphora from drooping and relaxation of the lower eyelid

3- Mouth

- Can't close the lips firmly → can't whistle or blow
- Buccinator is paralysed → food collects between cheek & teeth

4- Tongue

- Lesion proximal to entrance of chorda tympani (**6 mm** above the stylomastoid foramen) → loss of taste in anterior 2/3 of tongue



Treatment

A) Conservative

- Indication: Bell's palsy
- Method: 1- Electrical treatment & massage → maintain health of the muscle & prevent atrophy.
- 2- Active facial movements in front of a mirror several times daily
- Recovery begins after **3-4 weeks** & completed in **3-6 months**

B) Operative

- Indication: neurotmesis
- Method: 1- If the nerve is accidentally divided → nerve suture & if the gap is wide → nerve graft using the great auricular nerve
- 2- If repair is impossible → hypoglossal anastomosis (hypoglossal nerve is divided and its proximal end is anastomosed to the distal end of the injured facial nerve)
- 3- In late cases, face asymmetry improved by plastic operation

Hypoglossal nerve usually escapes injury in fractures of the base of skull since the anterior condyloid foramen is protected by a bony ridge → deflects the fracture towards the foramen magnum

Accessory Nerve injury

- Etiology: 1- Commonly during operations on the neck, particularly for removal of tuberculous or malignant lymph nodes
- 2- Fractures of the posterior fossa involving jugular

- Clinical picture:

- 1- Division in the anterior triangle → partial paralysis of trapezius & sternomastoid muscles (receive an additional nerve supply from the cervical plexus)
- 2- Division in the posterior triangle → the trapezius alone is affected → drooping of the shoulder, tilting of the scapula & inability to abduct the arms above the right angle due to imperfect scapula fixation



Hypoglossal nerve injury

- Etiology: 1- During operations for removal of tuberculous LNs or submandibular sialadenectomy operation
- 2- May be involved by malignant diseases.

- Clinical picture: hemiatrophy of the tongue → directed towards the paralysed side on protrusion



Brachial plexus injury

Etiology

1. Pressure injuries

Fractures of clavicle & shoulder dislocations or attempts to reduce them → injury to the medial cord of plexus

2. Open injuries (rare)

-By gunshot and stabs in the lower part of the posterior triangle or during axillary block dissection

3. Traction injuries

- Birth-injury (in vertex or breech presentations) from forcible stretching of the head during delivery
- Hyperabduction of the arm under anaesthesia → damage of 8th cervical and 1st thoracic nerves
- Forcible shoulder depression as when heavy weights fall on shoulder → damage to 5th and 6th cervical nerves

Clinical picture

1. Whole plexus type

- Motor: whole arm is paralysed
- Sensory: anaesthesia of upper limb except the medial side of the arm down to the elbow (intercostobrachial nerve) and for a more limited area on the outer side (supraclavicular nerves).
- Horner syndrome: ptosis, myosis, enophthalmos and anhidrosis

2. Upper-arm type (Erb-Duchenne paralysis) (C5, C6)

- Motor: **policeman or waiter's tip position** → limb hangs by the side with the forearm extended and pronated
- Sensory: anaesthesia over the outer side of the arm (Absent if 5th nerve only is involved)

3. Lower-arm type (Klumpke's paralysis) (C8, T1)

- Motor: 1- paralysis of the flexors of the wrist and fingers
2- Paralysis of intrinsic muscles of the hand
3- Claw-hand
- Sensory: Anaesthesia along the inner side of the forearm and the inner one and half fingers
- Homer's syndrome → indicates high lesion & bad prognosis.

Treatment

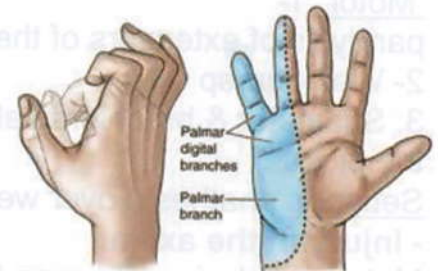
A) Conservative treatment

- Indication: closed injuries
- Method: 1. The arm is splinted in right-angled abduction → relax the deltoid and spinati and the forearm being flexed and supinated → to relax the biceps, brachialis and supinator
- 2. Elevation and massage → control edema
- 3. Active & passive movements of all joints → prevent stiffness
- 4. Electrotherapy → maintain tone and nutrition of muscles.

B) Surgical treatment

- Indication: degenerative lesions which fail to recover within **2 months**, particularly when neuroma is palpable
- Operation: remove scar tissue and the nerve ends are freshened, identified and sutured together.
- Prognosis: results are often disappointing

Whole plexus injury is rare since injury sufficient to damage all roots of the plexus is fatal



Neurapraxia recovers within **3 - 4 weeks** but axonotmesis takes very long time and physiotherapy should be continued for **2 years**

Radial Nerve injury

Etiology

- In the axilla → pressure by crutches or fractures & dislocations of the upper end of the humerus or attempts at their reduction
- In the spiral groove:
 - a. Fractures of the shaft of the humerus,
 - b. Falling asleep with the arm lying across the edge of a chair or table (**Saturday-night paralysis**).
 - c. Prolonged application of a tourniquet.
 - d. Outstretched arm rests on edge of the table during operations
 - b. Intramuscular injections of irritant drugs.

Clinical picture

-Injury at head of radius:

Motor: Paralysis of extensors of the fingers and thumb (**finger drop**)
(If wrist and proximal phalanges are supported & extended → middle and distal phalanges can be straightened by the action of the interossei and lumbricals)

Sensory: no sensory loss

- Injury in spiral groove:

Motor: 1-

paralysis of extensors of the wrist (**Wrist-drop**)

2- Weak grasp

3. Supinator & brachioradialis (supination can still be performed by the biceps)

Sensory: anathesia over web space

- Injury in the axilla:

Motor: paralysis of Triceps & anconeus (The forearm can only be extended by its own weight)

Sensory: back of the arm

Treatment

1. Conservative treatment

- a) Cock-up splint → slight hyperextension of hand
- b) Massage of forearm & electrical treatment

2. Nerve repair

-Indication: no recovery within **6 weeks**

- Prognosis: good because it is purely motor and because it controls coarse unskilled movements

- Recovery: expected in 9-12 months

3. Tendon transplantation (if nerve repair failed)

- a. Pronator teres to extensor carpiradialis for wrist extension
- b. Flexor carpiradialis to extensor digitorum for fingers extension
- c. Palmaris longus to extensor pollicis longus for thumb extension



Circumflex Nerve injury:

- Etiology:

1. Gunshot wounds
2. Direct blows on shoulder
3. Fractures of surgical neck of the humerus.
4. Dislocations of the shoulder.
5. Crutch palsy

- Clinical features:

1. Deltoid muscle is paralyzed and wastes → acromion becomes prominent & the shoulder flattened
2. The patient is unable to raise the arm from the side
3. Temporary anaesthesia over the posterior fold of the axilla

- Treatment:

Remove the cause of pressure then do splintage, massage and electric stimulation

Median Nerve injury

Etiology

Above the elbow: by tourniquets

At the elbow: 1- fractures of the lower end of the humerus

2- Dislocations of the elbow joint

3. Gunshot

AT the wrist: 1-cut wounds especially by glass due to bursting of bottles or thrusting the hand through a window

2- Carpal tunnel syndrome (Compression)

Clinical picture

Motor:

Injury at elbow → Paralysis of

1. The 2 pronators → loss of forearm pronation

2. Flexor carpiradialis → defective flexion of the wrist on the radial side and impaired radial abduction

3. Palmaris longus → weak hand grasp, especially on radial side.

4. The flexor pollicis longus → terminal phalanx of the thumb cannot

5. Flexor sublimis, outer 1/2 of flexor profundus & lateral lumbrical muscle → **pointing index**

Injury at the wrist → paralysis of

1. Short muscles of thumb (abductor pollicis brevis, opponens pollicis and flexor pollicis brevis) → thenar eminence is wasted & flattened and the thumb is extended by the side of the fingers →

simian or ape-like hand

2. Opponens → cannot raise the thumb forwards at right angles

3. Abductor pollicis brevis → **pen touching test**

4. There is paralysis of the outer 2 lumbricals.

Sensory: Anathesia over:

a) Lateral 2/3 of palm

b) Lateral 3 & 1/2

Trophic changes

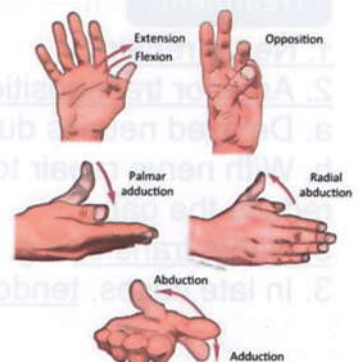
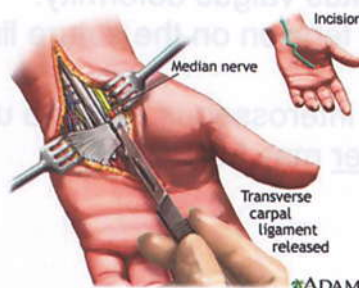
1- Tips of fingers are shrunken and tapering and cigarette burns are often seen on the anaesthetic fingers.

2- Severe causalgia in partial injuries

Treatment

1- Conservative treatment → splintage in position of function

2- Operative treatment → Division of flexor retinaculum for carpal tunnel syndrome



Ulnar Nerve injury

Etiology

1. Wounds, fractures and dislocations in the region of the elbow.
(Delayed ulnar neuritis often follows cubitus valgus deformity due to old injury of the lower end of humerus)
2. Wounds at the wrist

Clinical picture

Injury at the wrist:

Motor: Paralysis of

1. Medial two lumbricals → ulnar claw- hand (flexion of interphalangeal joints and hyperextension of the metacarpophalangeal joints of the medial two fingers)
2. Dorsal interossei → wasting with flattening of the hypothenar and interosseous spaces
3. Interosseous muscles → Inability to abduct & adduct fingers → patient cannot grip a sheet of paper between extended fingers (**card-board test**).

4. Adductor pollicis → **Froment's sign** (If the patient pinches a piece of paper between the thumb and fingers → terminal phalanx of the thumb is flexed by flexor pollicis longus)

Sensory: Anesthesia over the inner one and half fingers and ulnar 1/3 of hand **anteriorly only** since the dorsal cutaneous branch of the ulnar arises about **2-inches** above the wrist

Injury at the elbow:

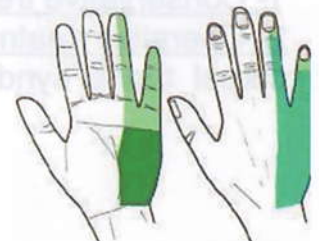
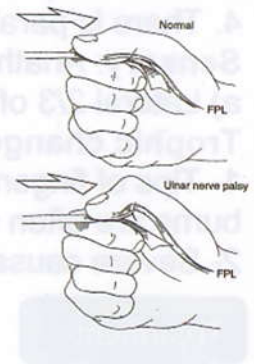
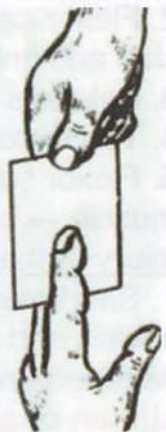
Motor: paralysis of

1. Flexor carpi-ulnaris → weakness in flexion & flattening of the inner border of the forearm.
2. The inner half of the flexor digitorum profundus → ulnar paradox (less clawing of medial two fingers compared to injury at wrist)
3. Small muscles of the hand with the exception of thenar muscles and the outer 2 lumbricals

Sensory: as above + dorsal 1/3 of hand

Treatment

1. Nerve repair
2. Anterior transposition of the nerve is indicated for:
 - a. Delayed neuritis due to cubitus valgus deformity.
 - b. With nerve repair to reduce tension on the suture line or to reduce the gap
3. Nerve transfer e.g. anterior interosseous nerve to ulnar nerve.
3. In late cases, tendon transfer may be useful.



Sciatic Nerve injury

Etiology

1. Deep wounds of the buttock and thigh.
2. Dislocations of the hip
3. Fractures of the pelvis.
4. Intramuscular injection of drugs

Clinical picture

Motor

- 1- Paralysis of all muscles below the knee → foot drop
- 2- If the nerve is damaged near its exit from the pelvis → paralysis of hamstrings (some degree of knee flexion is possible by action of the sartorius and gracilis muscles)

Sensory:

Anaesthesia of the leg and foot except the medial aspect of both (supplied by the saphenous nerve)

Trophic changes

- 1- Common on the sole of the foot and toes
- 2- Causalgia in the sole (if the lesion is partial)

Treatment

1. Conservative treatment

- a) Suitable support for deformities → prevent foot drop
- b) Massage and electrotherapy

2. Nerve repair

- A gap can be bridged by flexing the knee & extending the hip and after operation the knee is kept flexed for 3 weeks then gently and gradually extended

3. Orthopaedic treatment

- In persistent paralysis → suitable hinged knee-splint and by arthrodesis of the ankle or tendon fixation → great gait improvement

Differential diagnosis of complete claw hand:

1. Klumpke's palsy
2. Combined ulnar and median nerve injury
3. Volkmann's ischemic contracture
4. Dupuytren's contracture
5. Post burn scar contracture

Leg amputation is indicated only for persistent severe pain or extensive trophic ulceration

Common peroneal nerve injury

Etiology

1. Deep wounds of thigh
2. Fractures of the upper end of fibula
3. Subcutaneous tendotomy of the biceps tendon.
4. Pressure by strapping, bandages, splints and plasters.

Clinical picture

Sensory: Anaesthesia of dorsum of the foot and the front and outer side of the lower 2/3 of the leg

Motor: Paralysis of the extensor and peroneal muscles:

- a) In the early stages → foot drop
- b) Later on contraction of the opposing muscle → paralytic talipes equinovarus

Nerve injuries which may occur under anesthesia

- 1- Faulty injection or extravasation of nerve irritants as thiopental or propofol
- 2- Brachial plexus injury when arm is suspended at an angle $> 50^\circ$ to the operating table with external rotation → overstretch of the roots (Similar effect when allowing arm to drop off operating table, arm overextension with the head tilted to the opposite side or plexus compression by shoulder bracelets in Trendelenburgh position)
- 3- Ulnar nerve injury
 - When the arms are kept pronated by the side of the patient → nerve compression between the hard edge of the operating table and the medial epicondyle.
- 4- Radial nerve injury
 - a) Compression between the hard edge of the operating table & the humerus in the spiral groove.
 - b) Tourniquet injury and faulty I.M. injections in the arm
- 5- The sciatic nerve injury : a) I.M injections
 - b) Compression in the lateral position of hip surgeries
- 6- The common peroneal nerve
 - Compressed between the operating table & the fibula
- 7- The tibial nerve
 - Compressed in popliteal fossa by knee support in lithotomy position

Autonomic Nervous System

A) Parasympathetic system: originate from

1. Cranial outflow: pass through →

- a) The oculomotor nerve to the pupil → pupil constriction
- b) The facial nerve to the salivary glands → saliva secretion
- c) The vagus to the heart, lungs, alimentary canal, liver & pancreas → secretory and motor to the alimentary canal & inhibitory to heart

2. Sacral outflow

- **Course:** Pass via the 2nd, 3rd & 4th sacral nerves to form pelvic splanchnic nerve which lies close to the presacral nerve and runs upwards to the inferior mesenteric artery at its origin from the aorta.

- **Function:** concerned with the emptying → motor impulses to the rectum and bladder and causes penile erection.

B) Sympathetic system

1. Preganglionic fibres (white rami communicants)

- Are the axons of the cells of lateral horns of the grey matter of all the thoracic and upper 2 or 3 lumbar segments of the cord → pass from anterior roots to join the sympathetic chains
- They may form synapses in the nearest ganglion, pass up and down the chain to higher or lower ganglia, or pass through the ganglia to reach visceral ganglia and plexuses.
- Some Preganglionic fibres pass directly to medulla of adrenal gland which represents the synapse and postganglionic fibre.

2. Sympathetic chains

- These are a pair of ganglionated trunks that lie on the side of the bodies of the vertebrae and extend from base of skull to the coccyx.
- Each chain is formed of ganglia and fibres.
- There are 3 cervical ganglia, 11 or 12 thoracic, 4 lumbar and 4 sacral and the cords join over the coccyx to form the ganglion impar
- The inferior cervical & 1st thoracic ganglia are connected together to form **the stellate ganglion** (lies in a groove on the neck of 1st rib)

3. Visceral ganglia and plexuses

- Contain the cells whose axons pass direct to abdominal viscera.
- Their preganglionic fibres pass, without synapsing
- Main visceral ganglia are coeliac, superior & inferior mesenteric

4. Postganglionic fibres

- Are non-medullated fibres which arise from cells of ganglionated trunk and visceral ganglia and pass to the skin vessels and viscera
- Two definite bundles known as the grey rami communicantes, proceed from the ganglia of the trunk to each spinal nerve

Sympathectomy

Indications

1. Vascular conditions

- **Effect:** interrupts the vasomotor fibres → blood vessels dilatation and improvement of the local circulation

-Used in:

- a. Raynaud's disease → Pain is completely relieved but attacks of cyanosis may still occur.
- b. Buerger's disease → relieves the pain and promotes healing of ulcers (relapses after **2 or 3 year** are common)

2. Hyperhidrosis (Excessive sweating of the hands)

- **Operation:** cervico-dorsal Sympathectomy
(That of the feet responds to lumbar Sympathectomy)

Principles of cervico-dorsal Sympathectomy

- Sympathetic supply of UL is derived from 2nd & 3rd thoracic nerves.
- The preganglionic fibres enter the corresponding ganglia & ascend in the sympathetic trunk to synapse in the first thoracic ganglion (a few synapse in the 2nd ganglion from which the postganglionic fibres proceed in the nerve of Kuntz to the 1st thoracic nerve).
- To denervate upper limb → excise the 2nd & 3rd thoracic ganglia
- Sympathetic trunk below the 3rd ganglion is divided together with rami communicantes of the 2nd & 3rd ganglia & the nerve of Kuntz.
- The stellate ganglion is left intact to avoid Homer's syndrome.
- **Approaches:** A) Axillary approach B) Thoracoscopy
C) Anterior approach by transverse incision in lower part of neck

Principles of lumbar Sympathectomy

- Preganglionic fibres for the lower limb pass through lower thoracic & upper two lumbar nerves to the corresponding ganglia of the sympathetic trunk & descend in the trunk to synapse in the lower lumbar and sacral ganglia.
- Excision of lumbar chain → preganglionic denervation of lower limb
- **Operation:** the upper 3 lumbar ganglia are excised
- If bilateral Sympathectomy is required → preserve the 1st lumbar ganglion on one side, otherwise failure of ejaculation occurs

Nerve Tumours

Generalized neurofibromatosis (von Recklinghausen's disease)

Pathology

Site: widespread affection of the cerebrospinal nerves, nerve roots within the cranial cavity & spinal canal and nerve fibres in the skin, muscles and bones.

Grossly: affected nerves are diffusely and irregularly thickened with the formation of tumour-like swellings

Histologically:

- Proliferation of cells of endoneurium (perineurium & epineurium are unaffected)
- Composition: connective tissue fibres arranged in strands, bundles and whorls
- The nuclei are elongated, hyperchromatic & arranged in rows → palisade appearance.
- Nerve fibres may traverse the substance of tumour or may be displaced to one side but they aren't the seat of degeneration → no sensory or motor changes are observed

Clinical picture

Type of patient: often familial and begins in adolescence

Symptoms: 1- Commonly preceded with localized areas of pigmentation in skin (**cafe-au-lait patches**)

2- Slowly growing swellings

3- In malignant change → pain, anaesthesia and paralysis in the area of affected nerve with progressive increase in size of lesion

Signs: 1- Multiple variable sized fusiform tumours all over the body surface with the long diameter in the axis of the affected nerve

2- Lying in relation to the plexuses at the root of the limbs

3- Firm or soft in consistency

4- Mobile in a lateral direction but not in the line of the nerve.

5- The affected nerves may or may not be palpably thickened but painless

Neuro-fibromatosis:

-Definition:

proliferative condition of the endoneurium of nerves associated with tumour formation and sometimes with pigmentation of skin

- Include several varieties (either singly or Combination)

Solitary neurofibroma:

- Incidence:

- a) Between **20 and 50 years**
- b) Usually affect nerves of UL but may occur in spine, mediastinum or viscera.

-C/P: small elongated firm tender swelling very liable to cystic degeneration.

- Treatment:

Complete excision

Treatment

- Excision of all tumours is impossible
- Indication for operation: very large tumours, painful tumours and tumours producing pressure symptoms.
- The tumors should be completely resected.

Cutaneous neurofibromatosis (Molluscum fibrosum):

- Pathology: Multiple soft fibrous swellings arise in connection with the terminal filaments of the cutaneous nerves
- Size: vary from a pin's head to a lemon size or larger
- Shape: sessile or pedunculated
- Site: over the scalp, limbs, abdomen & back (palms of the hands and soles of the feet escape)
- In most cases, skin is the seat of pigmented areas (cafe-au-lait patches) or frank melanoma
- Treatment: excise large & unsightly tumours

Plexiform Neurofibroma

- Pathology: diffuse fibromatous thickening of branches of a nerve forming a beaded swelling under the skin
- Site: S.C. tissue of head & neck, large nerves of limbs & autonomic plexuses of the abdomen.
- Clinically: 1) The overlying skin is often pigmented and thickened and may hang down in pendulous folds (**pachydermatocele**)
- 2) > 1/2 of cases has manifestation of generalized neurofibromatosis

Elephantiasis Neuromatosa

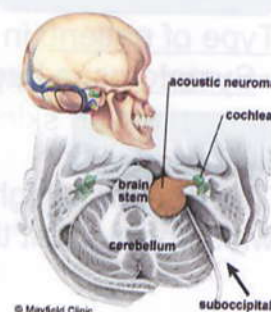
- Related to and may co-exist with cutaneous neurofibromatosis
- Type of patient: appear in childhood
- Site: affects one of the extremities, especially the lower
- Clinically: 1) affected part gradually increases in size
- 2) The skin & S.C tissues are greatly thickened
- 3) A variable number of tumours may be present and the skin may be pigmented and hairy

Acoustic Neuroma

- Incidence: 1) rarely associated with neurofibromatosis
- 2) Has familial incidence → dominant trait
- Often associated with tumours of the dura and choroid plexus.

Neurofibrosarcoma

- Origin: de novo in an apparently normal nerve or by malignant degeneration in a neurofibroma.
- Shape: of a spindle-cell or myxosarcomatous character
- Clinically: a painful rapidly growing tumour which invades surrounding tissues → anaesthesia, paralysis & distant metastases
- Treatment: a) In localized tumors → Wide excision
- b) In extensive tumours & post-operative recurrences → amputation



Ganglioneuroma

Pathology

- **Nature:** A benign tumour
- **Origin:** sympathetic trunks or adrenal medulla.
- **Histologically:** uni-or multipolar ganglion cells scattered among non-medullated nerve fibres
- **Grossly:** well-encapsulated
- **Size:** varies from a pea to a large melon,

Clinical picture

- **Type of patient:** in childhood
- **Symptoms:** a rounded or lobulated soft swelling which attracts attention by its size or pressure effects.

Treatment

Enucleation (easy since it is well-encapsulated)

Neuroblastoma

Pathology

- **Nature:** highly malignant tumour
- **Origin:** sympathetic chain or the adrenal medulla
- **Histologically:** composed of small round cells with hyperchromatic nuclei arranged diffusely or in rosettes (Ganglion cells and nerve fibrils are often present)
- **Grossly:** a reddish purple soft swelling liable to haemorrhage & degeneration that grows rapidly
- **Size:** reaches a large size and metastasizes by blood & Lymph vessels
- **Site:** most common in the abdomen and neck.

Clinical picture

- **Type of patient:** in early childhood
- **Symptoms:** a large retroperitoneal mass (soon metastasize)

Treatment

The tumour is highly radiosensitive but metastases are so early and wide spread that the prognosis is not favourable.

Surgery of the spine and spinal cord

Spina bifida

Definition

Split spine

Prevention

Folic acid administration during pregnancy

Site

Mostly the lumbar spine

Types

1- Spina bifida occulta (10% of population)

- Defect: complete development except for a bifid spine

- Skin is connected to meninges by a fibrous band (**membrana reuniens**)

- Clinically: lipomatous swelling, skin dimple or a tuft of hair

- Complications: In a minority of cases, at puberty → incontinence of urine or lower limb motor deficits (because spine elongates faster than the spinal cord → membrana reuniens causes cord traction)

- Investigation: Plain X-ray → deficient laminae of spine

2- Meningocele

- Definition: bulging of meninges only through the spinal defect

3- Meningomyelocele

- Definition: bulging of meninges & neural tissue (normally spinal cord or cauda equina) within the sac → dark shadows on transillumination

- Incidence: commonest type of spina bifida

- Association: Arnold Chiari malformation (4th ventricle lie at a level below foramen magnum → obstruct CSF flow → hydrocephalus)

- Prognosis: if untreated **75%** die within 1st year of life.

4- Syringomyelocele (rare)

- Definition: dilatation of central canal

5- Myelocele (complete spina bifida)

- Defect: failure of fusion of the neural tube

- Clinically: open spinal plate → dribbles CSF (incompatible with life)

Spondylo- listhesis

- Definition:

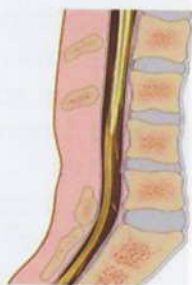
disorder in which there is 4th or 5th

lumbar vertebral defect → its body slip forwards on the sacrum

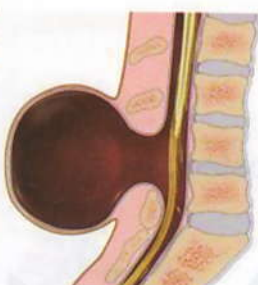
- C/P: may be clinically obvious if there is a step in the lumbosacral spine

- Treatment:

- In early cases → physiotherapy, rest and spinal support
- In gross cases → spinal fusion by bone grafting with sometimes removal of a prolapsed intervertebral disc



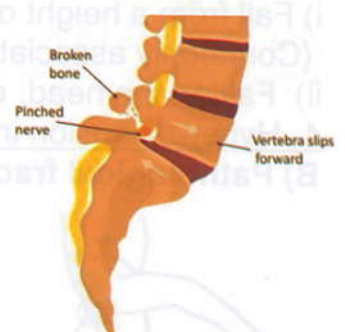
Spina bifida occulta



Meningocele



Myelomeningocele



Clinical picture

1- Antenatal screening

- i) Ultrasound.
- ii) High levels of alpha-fetoprotein in amniotic fluid.

2- Spina bifida occulta

- i) Usually asymptomatic → accidental finding in X-rays
- ii) Lipoma, skin dimple or a tuft of hair over the bifid spine.
- iii) Rarely urinary incontinence starting at adolescence.

3- Meningocele

- i) No neurological manifestations.
- ii) Cystic translucent swelling with an expansible impulse on cough
- iii) The swelling is compressible

4- Meningomyelocele and syringomyelocele

- i) Paraplegia, wasting or contractures with sensory loss
- ii) Marked Trophic changes (particularly perforating foot ulcers)
- iii) Commonly associated with hydrocephalus

Treatment

1- Spina bifida occulta

- No treatment required
- If membrana reuniens produces incontinence → surgical division

2- In other forms → surgery (early as possible)

- a) Tile operation → cover the defect by flaps from the lumbar fascia (Excision is done only in pure meningocele & not in types which contain nerve tissue)
- b) Ventriculo-peritoneal (or atrial) shunt for hydrocephalus

The order of frequency of spina bifida types:

1. Spina bifida occulta (usually asymptomatic)
 2. Meningo-myelocele (neurological manifestations + lumbar swelling)
 3. Meningocele (lumbar swelling)
 - Spina bifida may present as isolated anomaly or associated with other anomalies (**VACTERL**)
- V**ertebral
Anorectal
Cardiac
Tracheal
Esophageal
Renal
Lumbar anomalies

Fractures and dislocations of the spine

Etiology

A) Trauma

1- Hyper-flexion (most frequent type)

- as in RTA & fall of a heavy object on the back of the head

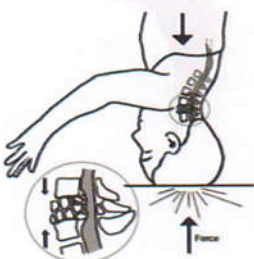
2- Hyperflexion and rotation

3- Vertical compression trauma

- i) Fall from a height on the feet or the buttocks (Commonly associated with calcaneus fractures)
- ii) Fall on the head, e.g., diving in shallow water.

4- Hyper-extension injuries (uncommon)

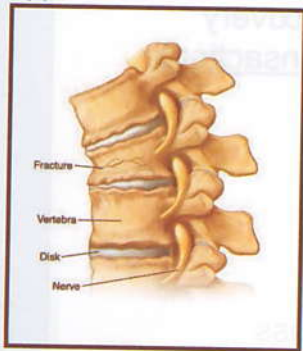
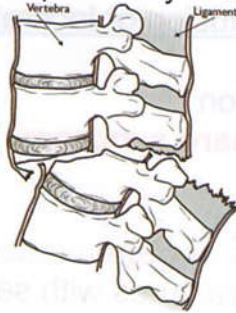
B) Pathological fractures due to metastases or osteoporosis



- Fractures & dislocations of vertebrae may produce spinal cord & nerve root injuries.
- Site: mostly at junction of mobile & rigid parts in lower cervical and dorsolumbar regions

Types

A) According to morphology

	1- Wedge compression fracture	2- Fracture dislocation	3- Comminuted (burst) fracture
Trauma	Hyper-flexion 	hyper-flexion accompanied by rotation 	Vertical compression 
Body	Front compression	Forward dislocation of vertebra with articular processes breaks	Comminuted
Ligaments & facets	The posterior spinal ligaments are intact (stable)	The posterior ligaments are also torn (unstable)	Intact (stable)
Spinal cord or caudal damage	undamaged	Commonly damaged	Sometimes damaged

4- Pure Dislocation

- Occur in cervical spine only because its articular processes are rather horizontal → similar consequences to fracture dislocation.

5- Avulsion fractures of transverse and spinous processes

- Not accompanied by neurological injury
- No treatment required

B) According to stability

1- Stable fractures: wedge compression, comminuted (burst), and avulsion fractures of the transverse & spinous processes.

2- Unstable fractures: fracture dislocations & pure dislocations

Neurological injury

- Pathogenesis: injury of spinal cord, nerve roots or both

- Effect: Paraplegia or quadriplegia

- Prognosis: depends on level & nature of the

- Level of lesion:

1. Injuries at level of D12 or L1 vertebra → combined cord & root damage → transection of sacral cord & paralysis of muscles controlling the hip

2- Injury at level of D10 → isolates the entire lumbar and sacral cord → permanent paralysis of the lower limbs and viscera.

- Dislocations and fracture dislocations of atlas and axis vertebrae are serious → cord transection above the level of respiratory muscles
Innervation → **fatal**

- **Hangman's fracture** → Atlas dislocation with or without odontoid process fracture

3- In cervical spine fractures

- Level of cord injury corresponds to the level of bone injury
- High cervical cord transaction is fatal because all respiratory muscles are paralyzed

- Nature of the lesion:

1- Spinal cord concussion

Like cerebral & nerve concussion (neurapraxia) → recovery

2- Spinal cord damage by contusion or laceration (transaction)

- Do not recover.
- Types: i) Complete transaction
- ii) Hemisection (**Brown-Sequard syndrome**)

3- Cauda equine injury

-Stages of cord transaction:

1- Shock stage

- Timing: An early stage
- Presentation: complete flaccid paralysis below the level of injury, loss of tendon reflexes and an atonic distended bladder.
- Duration: lasts for a few days
- During this stage, cord transaction is suspected with the following:
 - i) Complete loss of all forms of sensation below level of lesion
 - ii) If sensory loss & paralysis rise after injury → ascending edema
 - iii) A damaged cord, which has not recovered in 48 hours, will never recover
 - iv) A very sure sign of cord transaction is the appearance of the mass reflex (**at 3-6 weeks**) and the presence of anal and penile reflexes in the absence of sensation in the legs.

2- Stage of reflex activity

- Presentation: reflex function below level of transaction recovers → spastic paralysis and reflex emptying of the bladder.

Clinical picture

- Spine injury is suspected after any major trauma.
- A cervical collar is applied & immobilized the head
- Search for other injuries of the skull, chest, abdomen or limbs
- Local signs: (patient is gently rolled onto his side in one piece)
 - 1- A slight kyphosis may be visible or felt.
 - 2- Local-tenderness over the spinous process.
 - 3- No gap is palpable between the spinous processes in stable fracture. In an unstable fracture, separation of the spinous processes may be evident.
 - 4- No attempt to test back movements → paraplegia
- Neurological assessment
 - 1- Motor power
 - 2- Sensations
 - 3- Reflexes

- The spinal cord ends at lower border of 1st lumbar vertebra, fractures below this level → root lesions of cauda equina
- The first sacral cord segment is opposite the dorsolumbar junction
- The first lumbar cord segment in adult is at the level of D10 vertebra
- In cervical spine → cord segments correspond to the level of the vertebrae
- In concussion, joint position which ascends in the posterior column is preserved
- The stability of spine fracture depends on the integrity of the posterior ligaments
 - Always suspect spine fractures in victims of major trauma

Investigations

1- Plain X-ray

- **Views:** A-P, lateral views & a special view through the open mouth
→ diagnose fractures and dislocations of the atlas vertebra.

- Cervical spine films should show all seven cervical vertebrae & T1

2- CT scanning (**most sensitive**)

3- MRI (best to reveal spinal cord injuries)

Treatment

A) Initial management

- Treat first life-threatening problems as airway obstruction, breathing and bleeding deserve priority in management

- Prevent spine movement by:

1- Apply cervical collar to immobilize the head during transport and examination

2- The trauma victim is moved on to the stretcher all in one piece.

(**Never lifted by the shoulders and the thighs**)

- In hospital, the patient is nursed on a bed with fracture boards.

- Treat shock

B) Stable fractures

1- Wedge compression fracture

- Extension exercises for the back muscles.

- Prognosis: Within a month, he is allowed up & can resume work in

3 months with a painless, mobile and powerful back

- The compressed vertebra remains wedge shaped.

2- Comminuted fracture.

- Apply plaster jacket in the neutral position (straight spine) for **3 months** & patient can walk with it

- Regular spinal exercises after applying the plaster jacket

- Prognosis: severe pain and delayed healing

3- Avulsion fractures of the transverse or spinous processes

- Rest and analgesics.

C) Unstable fractures

1- Traction

2- Hard cervical collar



Pott's disease of the spine

Clinical picture

Symptoms

- General: night fever & sweat and loss of weight & appetite
- Local: pain which increases on movement may be referred along the nerves to the abdomen.

Signs

- 1- Limitation of movements in all directions (**most important sign**)
→ child is unable to pick a coin from the ground with knees straight, he has to bend his knees and on rising he climbs on himself.
- In cervical region, patient supports his head with his hands and rotates as a whole
- 2- Angular kyphosis (if thoracic region affected)
- In cervical & lumbar regions → masked by normal lordosis → just obliterated & spine looks straight
- 3- Cold abscess

Investigations

1- Plain X-ray

- a) Two or more vertebrae are affected.
- b) Intervertebral disc space is narrowed then completely lost.
- c) Adjacent surfaces of the vertebrae are irregular.
- d) Absence of new bone formation (**characteristic**)
- e) Paravertebral abscess shadow

2 -CT scan

Vertebral bodies may be collapsed with areas of bone destruction

Treatment

A) Conservative treatment (Mainly)

- 1- Anti-tuberculous medications for **9 months**.
 - 2- Rest and good nutrition.
 - 3- Spine support by plaster cast for a short period then by spine brace after improvement
 - 4- Periodic clinical, hematologic and radiographic assessment.
- Prognosis: most cases are cured even with early paraplegia

B) Surgery

- Indications: Early Pott's paraplegia with

- 1- No sign of recovery after **3-4 weeks** of conservative treatment.
 - 2- Paraplegia getting worse in spite of conservative treatment.
- Aim: decompression of the spinal cord

- Paraplegia in Potts' disease

- Incidence:

- 10%** of cases
- The level of cord affection is usually in upper dorsal spine (narrowest part of spinal canal)

- Etiology:

compression on the spinal cord by:

- a) Cold abscess
- b) Angulation of spine,
- c) Thrombosis of the anterior spinal artery.



Intervertebral disc prolapse

Etiology

- 1- Sudden strain (80%) → the annulus ruptures → nucleus pulposus protrusion
- 2- Degeneration of the annulus (20%).

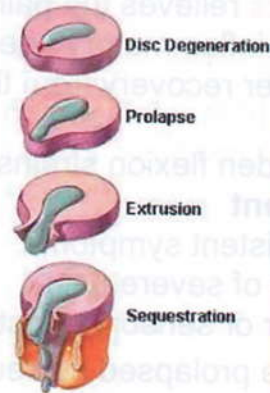
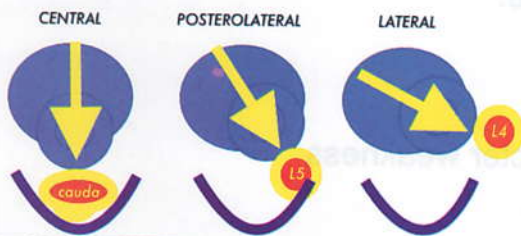
Level

In the most mobile segments of the spine

- 1) 80% in the lumbar region, particularly L4/L5 and L5/S1.
- 2) 19% in the cervical region, particularly C5/C6 and C6/C7.

Direction

- 1- Posterolateral → compress nerve roots
- 2- Posteriorly → compress the spinal cord
- 3- Into the vertebral body (**Schmorl's node**)



Intervertebral disc anatomy

- Composition:

central nucleus of a jelly like substance (**nucleus pulposus**) enclosed by a fibrous ring (**the annulus fibrosis**), the whole being enclosed between fibro cartilaginous plates above and below.

- Function:

- 1- Shock absorbent
- 2- Allow normal mobility between the adjacent vertebrae

Lumbar disc prolapse

Clinical picture

1- Low back pain

-Typically while lifting a heavy object → spinal muscles go into painful spasm → unable to straighten up.

2- Root pain

- **Etiology:** nerve roots compression
- **Radiation:** down the back of the lower limb
- **Increases by:** coughing.

3- Motor and sensory changes

a) L4/5 disc prolapse

Motor → weakness of ankle dorsiflexion

Sensory → Pain and anathesia of back of the thigh, lateral aspect of the leg and dorsum of the foot

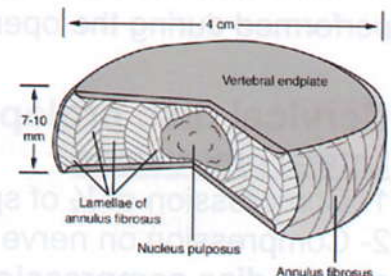
b) L5/S1 disc prolapse

Motor → weak planter flexion of ankle & absent ankle jerk

Sensory → Pain & anathesia of back of leg and sole of foot

4- A Scoliotic position & limping gait to reduce nerve root pressure

5- Straight leg raising or flexion of the spine → increases pain



Investigations

- 1- Plain X-ray of spine (PA and lateral views).
 - a) Disc space narrowing
 - b) In chronic cases → osteophytes at the margins of the vertebrae
 - c) To exclude bone diseases with similar manifestations
- 2- CT scan and MRI → accurately localize the site of disc prolapse



Treatment

A) Conservative treatment

- 1- Rest in a bed on boards placed under the mattress to give a rigid support for **2-4 weeks** relieves the pain in the majority of cases.
- 2- Analgesics & anti-inflammatory agent.
- 3- Physiotherapy after recovery from the acute attack.
- 4- Weight reduction.
- 5- Avoidance of sudden flexion strains on the spine.

B) Surgical treatment

- Indications:
- 1- Persistent symptoms.
 - 2- Recurrent attacks of severe pain.
 - 3- Evidence of motor or sensory affection or sphincter weakness
- Aim: removal of the prolapsed nucleus pulposus.

-Approach: either by

- 1- **Laminectomy** through a vertical midline incision in the back
- 2- **Microdiscectomy** by incising the tissues between adjacent laminae (can be done by endoscope)

- Causes of pain recurrence after surgery:

- 1- Removal of a disc from a wrong level.
- 2- Presence of another undiagnosed disc prolapse.
- 3- Presence of interstitial neuritis due to prolonged compression
- 4- Osteoarthritis of the intervertebral joints → spinal fusion should be performed during the operation for removal of the disc

Cervical disc prolapse

Lateral protrusions

- 1- Compression of $\frac{1}{2}$ of spinal cord → Brown Sequard syndrome
- 2- Compression on nerve roots depends upon the disc level

a) C5/6 disc compression

Motor → weakness of biceps & diminished biceps & supinator jerks
Sensory → Pain & anathesia over the outer border of the arm and dorsum of forearm

b) C6/7 disc compression

Motor → weakness of fingers extensors & diminished triceps jerk.
Sensory → Pain & anathesia over the back of upper arm & forearm
3- Movements of the spine aggravate the pain while rest and traction of the spine alleviate it

Midline protrusions

- 1- Compression of anterior part of the cord → quadriplegia
- 2- Compression of anterior spinal artery → affect pyramidal and spinothalamic tracts

Treatment

Conservative

- 1- A collar to limit movements of the neck.
- 2- In severe cases → traction by physiotherapist for about **20 minutes** every day
- 3- Analgesics

•If there is evidence of cord compression with developing quadriplegia & urinary incontinence → urgent decompression by laminectomy followed by spinal fusion

Spondylosis

Definition

A degenerative condition often a part of generalized osteoarthritis

Clinical picture

- 1- Pain in a nerve-root distribution.
- 2- Limitation of movement.
- 3- Stiffness.

Investigations

Plain x-ray:

- 1- Osteophytic lipping of the edges of vertebrae
- 2- Osteophytes particularly around the vertebral foramina
- 3- Narrow disc spaces

Many of the symptoms and signs of spondylosis are similar to disc prolapse
- An element of disc prolapse is a common feature of spondylosis

Treatment

(Similar to disc prolapse)

Conservative

- 1- Analgesics as anti-inflammatory agents
- 2- Mild cases → physiotherapy to strength spinal extensor muscles
- 3- Traction for varying periods may be valuable.
- 4- Spine support.
 - a) Lumbar spine → abdominal corset with steel stays or plaster jacket in a few severe cases
 - b) Cervical spine → cervical collar

Surgery

- Indications:
- 1- severe persistent symptoms
 - 2- Evidence of advancing nerve pressure

Operation: Laminectomy for decompression followed by spine fusion with bone grafts.



Spine tumors

Extradural tumours

Pathological types

- 1) Secondary deposits (**the commonest**)
 - **Source:** thyroid, breast, lung, kidney or prostate cancer
 - **Nature:** osteolytic except in cancer prostate → osteosclerotic
- 2) Sarcoma
- 3) Chordoma
- 4) Neurofibroma → may be "dumb-bell" in shape

Clinical features

- Symptoms and signs of irritation of nerve roots followed by pressure symptoms

Intradural extramedullary tumours

Pathological types

- The most important are meningiomas and schwannomas.

Clinical features

- 1- Sensory and motor weakness depending on tumor site
- 2- Symptoms due to pressure on the cord itself occur later, in hemisection compression → **Brown Sequard syndrome**

Intramedullary tumors

- Usually Gliomas or ependymomas.

Clinical features

- 1- Cord signs present first, while root pains tend to occur later.
- 2- Paralysis may occur on both sides (sometimes crossed anaesthesia on one side and paralysis & hyperesthesia on the other)
- 3- Dissociated sensory loss
- 4- Urinary incontinence (early)

Investigations

- 1- Plain X-ray of the spine → bone erosion or other bony abnormality in nearly **50%** of patients
- 2- CT scan and MRI

Treatment

Tumour removal and laminectomy → cord decompression

Most frequent site of:

- 1- Spondylolisthesis → L5/S1
- 2- Spina bifida → Lumbar
- 3- Fracture → Dorsolumbar and cervical
- 4- Pott's disease → Dorsolumbar
- 5- Disc prolapse → Lumbar (L4/L5 & L5/S) & Cervical (C5/C6 & C6/C7)

Brown Sequard syndrome

There is weakness and loss of vibration on the same side as the lesion and loss of pain on the opposite side.

Complications:
Paraplegia if pressure is not relieved



Cardiothoracic surgery

Chest injuries

Etiology and mechanism of injury

1- Blunt trauma → crushing of the chest wall

- Examples: a) blows to the chest wall b) FFH
c) Motor car accidents

2- Penetrating trauma

- Examples: by stabs or missiles
- The kinetic energy of the bullet is proportional to $(M \times v^2)$
- Damage caused by Gun shots > pistol shots > stabs.

3- Blasts

- The blast wave disrupts the alveolocapillary membrane → haemorrhage and leakage of interstitial fluids into the alveoli.
- Presentation: dyspnea and cough with frothy bloody sputum.
- In addition, the produced missiles from an explosion may cause blunt or penetrating injuries.

Consequences

- | | |
|-------------------|--|
| 1- Fractured ribs | 2- Cardiac injuries |
| 3- Haemothorax | 4- Injuries to the major blood vessels |
| 4- Pneumothorax | 5- Diaphragmatic injuries |
| 6- Lung injuries | 7- Oesophageal injuries |

Causes of respiratory insufficiency

- 1- Depression of respiratory centre due to head injury or narcotics
- 2- Airway obstruction by F.B. or secretions.
- 3- Unstable thoracic cage, e.g., flail chest.
- 4- Lung collapse due to haemothorax or pneumothorax.
- 5- Pulmonary contusion or laceration.
- 6- Diaphragmatic rupture.

Management

A) Follow the principles of ATLS (A, B, C, D, E).

B) In primary survey 5 major thoracic problems are life threatening:

- 1- **Tension pneumothorax** → Insert needle in 2nd intercostal space
- 2- **Open pneumothorax** → Seal the wound by an adhesive dressing which is fixed at three sides.
- 3- **Massive haemothorax** → Resuscitate then insert intercostal tube
- 4- **Flail chest** → Stabilize the flail segment by cotton gauze and compressive bandage.
- 5- **Cardiac Tamponade** → Pericardiocentesis (temporary relief)

- Incidence:
20% of all trauma victims
- Chest trauma especially within the "BOX" area (nipples to mid clavicular points) can be fatal in poly trauma if not prioritized & dealt with.
- Most chest injuries (blunt & penetrating), are managed by intercostal tube insertion. Thoracotomy is seldom (**10%**) indicated
- Shortness of breath with hypoxia is the most immediate threatening feature → interventions must ensure adequate ventilation and oxygenation.

- Proceed to 2nd survey and perform meticulous chest examination:

1- **Inspection**: for any wound and assess chest expansion.

2- **Palpation**: for tenderness, S.C. emphysema and assess the position of the trachea.

3- **Percussion**: the note may be impaired (**lung collapse**), dull (**haemothorax**), resonant (**pneumothorax**) or tympanitic (**tension pneumothorax**)

4- **Auscultation**: to assess the air entry

• A stab wound or a bullet injury to the lower chest may also cause an abdominal injury.

• In chest injuries, the most common cause of death is respiratory insufficiency which presents mainly by restlessness and agitation.

• The surest method to diagnose hypoxia is to do **ABG**.

Rib fractures

Etiology

1- Direct violence

- Examples: blow or crush

- Effect: a) Visceral injury (as the broken ends are driven inwards)
b) In severe crushes, several ribs may sustain double fractures → depression of the fragments & paradoxical movement (**flail chest**).

2- Indirect violence

- Examples: compression of chest → bends ribs beyond their natural elasticity → give way at sites of maximum stress (usually at angle)

- Visceral injury is uncommon (as broken ends are driven outwards)

3- Muscular violence

- Examples: during violent sneezing, coughing or lifting a heavy weight → may cause fracture of the anterior segment of a rib in old people (**senile osteoporosis**)

Simple rib fractures

- Clinically:

Symptoms: pain

Signs: tenderness & possibly crepitus at the fracture site.

- Complications:

1- Limitation of respiratory expansion by the pain → atelectasis & pneumonia

2- Undetected underlying haemothorax or pneumothorax (lethal)

3- Fractures of 1st or 2nd ribs → injury to subclavian vessels or brachial plexus

4- Fracture of lower ribs → liver or splenic injuries.

- Investigations: Plain chest X-ray → detect any air or fluid collection

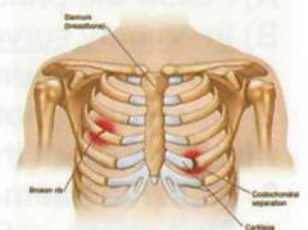
- Treatment:

1- Relief of pain to allow adequate lungs expansion

2- Strong analgesics as pethidine or NSAIDs

3- Intercostal nerve block or epidural analgesia (for persistent pain)

4- Chest physiotherapy, mucolytics and expectorants



Flail chest

- Definition: three or more ribs are fractured at two points due to a crushing injury of the chest

- Integrity of chest wall is lost & flail segment moves paradoxically

a) During inspiration → the flail segment retracts

b) On expiration → flail segment bulges

Effect: 1- limits the patient's ability to create enough -ve intrathoracic pressure to ventilate the lungs → interferes with respiratory efficiency and leads to sputum and CO₂ retention.

2- Side to side movement of the heart and mediastinum

(**Mediastinal flutter**) → interferes with venous return.

• Usually have underlying pulmonary contusion or laceration

-Treatment:

1- Relief of pain by strong analgesics, intercostal nerve block or thoracic epidural analgesia.

2- Initial stabilization by cotton gauze plus compressive bandage or elastic chest belt.

3- Encourage physiotherapy.

4- Endotracheal intubation and mechanical ventilation using PEEP is indicated in:

a) Combined head injury causing disturbed/lost consciousness

b) Tachypnea (respiratory rate **>35/min**).

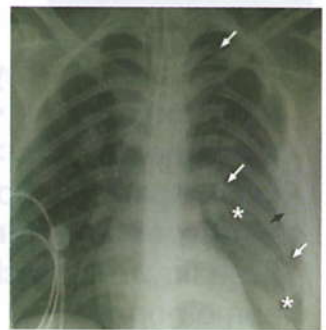
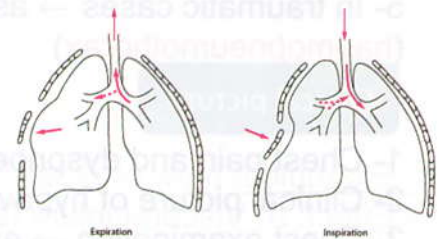
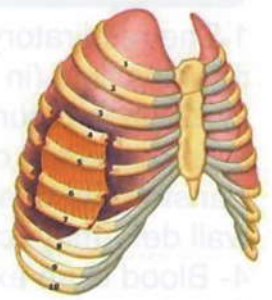
c) PaO₂ < **60** mm Hg.

d) PaCO₂ ≥ **50** mm Hg

e) Age **>50** years.

- This internal pneumatic fixation acts as a pneumatic mattress which supports the flail segment until provisional healing occurs.

• Internal fixation of fractured ribs by stainless steel wires or special nails is only done if there is an indication for thoracotomy



Haemothorax

Etiology

1-Traumatic

-Examples: closed or penetrating injuries

- Source of bleeding: intercostal or internal mammary vessels.

2- Post-operative

- Following cardiac, pulmonary and oesophageal operations.

- Or following insertion of a central venous line

3- Pathological

a) Tumours of the lungs, pleura or mediastinum.

b) Leaking thoracic aortic aneurysms.

Bleeding from lung laceration is not profuse because the pressure in the pulmonary circulation is low & when the lung is compressed by haemothorax → bleeding vessels are compressed.
- Only **10%** of cases bleeding is from the pulmonary circulation

Pathological sequelae

- 1- The respiratory & cardiac movements → defibrinate blood → so remains fluid (in some cases, blood may clot → difficult to aspirate)
- 2- Blood in pleural space is irritant → excites formation of effusion
- 3- In neglected cases → fibrin deposit on both layers of pleura → transformed to a fibrous layer (**fibrothorax**) → lung collapse & chest wall deformity (crowding of ribs)
- 4- Blood is an excellent culture medium and infection is common.
- 5- In traumatic cases → associated pneumothorax (**haemopneumothorax**)

Clinical picture

- 1- Chest pain and dyspnoea.
- 2- Clinical picture of hypovolaemia.
- 3- Chest examination → evidence of fluid in the pleural space:
 - a) Diminished lung expansion
 - b) dullness on percussion
- In massive haemothorax → mediastinum shifted to opposite side.

Investigations

1- Plain chest x-ray

- If haemothorax is < 500 ml → obliteration of costophrenic angle.
- If haemothorax is > 500 ml → an opacity rising to the axilla
- Haemopneumothorax → a transverse air-fluid level & lung collapse

2- Intercostal aspiration → reveals blood

- Free flow of non-clotting bright blood denotes excessive haemothorax

Treatment

- **Aim:** drain the blood as early as possible to allow full lung expansion and avoid the harmful effects of haemothorax.
- **Method:** 1- Insert a venous cannula and correct hypovolaemia.
- 2- If there is pain, an analgesic is prescribed.
- 3- Definitive treatment is by inserting a chest tube in the 5th intercostal space in the mid-axillary line then be connected to an underwater seal → drainage of blood and follow-up of its amount.
- **Signs of improvement:** 1- the patient is stable
- 2- The amount of drained blood is decreasing & lung is expanding
- Remove tube when no more drainage & lung is fully expanded

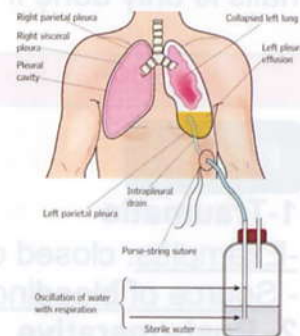
-Indications for thoracotomy

A) Early indications

- 1- Chest x-ray reveals complete opacification of the whole hemithorax or wide mediastinum > 8 cm.
- 2- Drainage of > 2000 ml of blood after insertion of the tube.
- 3- Bleeding > 200 ml/hour for 3 successive hours.
- 4- Associated with injuries, e.g., the oesophagus or cardiac tamponade.

Sternal fracture

- **Etiology:** 2^{ry} to deceleration injury to a car driver not wearing the seat belt → the steering wheel hits the sternum
- Its presence raises the suspicion of serious intrathoracic injuries as myocardial and pulmonary contusions, rupture of aorta & haemothorax



B) Delayed indications

- 1- Clotted or loculated haemothorax (diagnosed by failure of the tube to drain blood) → mini-thoracotomy for evacuation of blood clot
- 2- In the presence of foreign bodies.
- 3- Fibrothorax needs thoracotomy and decortication of the lung.

Pneumothorax

Etiology

1- Traumatic: blunt or penetrating injuries of the chest.

2- Spontaneous:

- a) Rupture of emphysematous bulla, solitary lung cyst or cystic lung.
- b) Rupture of a small subpleural tuberculous cavity. In this case, there is underlying pulmonary tuberculosis → hydropneumothorax

3- Iatrogenic:

- a) Positive pressure ventilation complicated by rupture of alveoli (**barotrauma**)
- b) During insertion of a central venous line.
- c) Injury to the pleura in upper abdominal operations

Types

I- Simple pneumothorax

- A limited amount of air is introduced in the pleural cavity.

- Presentation: chest pain and slight dyspnoea.

- Examination: diminished air entry & hyper resonance

(**No mediastinal shift**)

- Investigation: Plain chest x-ray → translucency, absence of lung markings & the edge of collapsed lung is usually visible

- Treatment:

- a) If the amount of air is small & no dyspnoea → conservative treatment until spontaneous absorption of the pneumothorax
- b) If the patient is dyspnoeic → an intercostal chest tube is inserted until expansion of the lung becomes complete

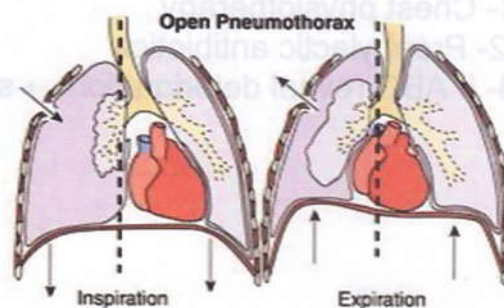
Open pneumothorax (sucking chest wound)

- Etiology: 2^{ry} to a wound in the chest wall → communication between the pleural space and the atmosphere.

- **During inspiration** → air enters into the pleural cavity through the opening → reducing intrathoracic negativity & reducing normal flow of air through the trachea to lungs (because the chest wall defect offers less resistance to flow)

- With large size hole → air completely stops flowing to the lung on the affected side

- The mediastinum shifts back & forth with each breath and cardiac output is reduced (**mediastinal flutter**)



-Diagnosis: Clinically by the sound of air going through the defect

-Treatment: (it is an emergency)

- Immediately apply an adhesive external dressing to stop movement of air through the defect

- Repair the wound in theatre and insert an intercostal chest tube to drain the plural air.

Tension pneumothorax

- Definition: A small wound of the chest wall involving visceral pleura or in a bronchus → a valvular opening that allows air to enter the pleural cavity during inspiration but prevents its exit during expiration → gradual lung collapse & mediastinum shifts to opposite side.

- Presentation: severe dyspnea due to compression of the opposite lung → respiratory arrest

- Examination: diminished air entry, hyper-resonance, shift of the trachea & mediastinum and engorged neck veins.

- Complications: obstructive shock (because it prevents cardiac Filling) → rapidly fatal

-Treatment:

- Immediately insert a wide bore needle in the 2nd intercostal space → decompression of the pneumothorax.

- Later, insert intercostal chest tube connected to an underwater seal (Continuous air bubbling through intercostal tube denotes possibility of a broncho-pleural fistula → thoracotomy needed to close it)

Tension pneumothorax is an emergency

→ no time for x-ray diagnosis



Lung injuries

-Include: lung haematoma, contusion, laceration or blast injuries

- Etiology: impact injuries to the chest wall

- Presentation: shortness of breath and haemoptysis.

- There may be haemothorax or pneumothorax.

- Suspected with high-energy trauma as RTA especially with sternal and multiple rib fractures

- Investigations:

1- Chest x-ray → opacity, haemothorax or pneumothorax

2- Arterial blood gases.

-Principles of treatment:

1- Chest physiotherapy

2- Prophylactic antibiotics

3- If ABG reveal deterioration → start mechanical ventilation



Cardiac injuries

Etiology

- 1- Penetrating injuries (90%): by stabs or bullets.
- 2- Blunt injuries: by blows to the sternum or sudden deceleration of unrestrained motor car driver

Possible consequences

- 1- Myocardial trauma → contusion with or without a mural thrombus (similar to M.I.) or lacerations → heal by fibrous tissue → aneurysm formation or heart failure (lacerations can cause cardiac tamponade)
- 2- Rarely, trauma causes rupture of valve or interventricular septum

Cardiac tamponade

- Definition: Accumulation of blood the pericardial sac → compresses the heart & impairs its relaxation and consequently, its filling

- The rate of blood accumulation is more dangerous than amount.
- 150 ml blood may be fatal if they rapidly accumulate

- Clinical features:

- 1- Dyspnea and irritability
 - 2- Unexplained sustained tachycardia
- 3- **Beck's Triad**:

- a) Blood pressure is persistently low despite IV fluids & blood transfusion
- b) Congested neck veins
- c) Weak or inaudible cardiac sounds.

- Investigations:

- 1- ECG → Low voltage, extra-systoles or A.F.
- 2- Plain chest x-ray → big cardiac silhouette or flask shaped heart (Absence of this feature does not exclude a tamponade)
- 3- FAST with subxiphoid pericardial window.
- 4- Needle pericardiocentesis

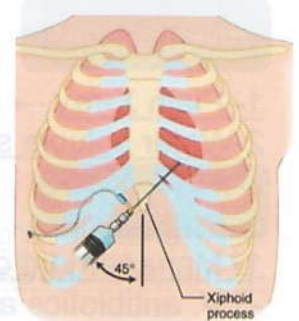
- Treatment:

1- For temporary relief → pericardiocentesis or subxiphoid pericardiotomy

- Pericardiocentesis a cannula is introduced in the subxiphoid area with 45° angle from the skin & 45° angle laterally towards the mid clavicle (better done under ultrasound guidance)

2- Definitive treatment → thoracotomy and pericardiotomy for repair of injury

Tension pneumothorax, massive cardiac contusion and cardiac tamponade cause cardiogenic shock
- Neck veins show characteristic Congestion in the presence of low blood pressure



Injuries to the major blood vessels

• Etiology: deceleration accidents → thoracic aorta may be injured at the junction of the arch and the descending part

• Investigations:

- 1- Plain chest x-ray → a wide mediastinum
- 2- CT angiography (**diagnostic**)



Diaphragmatic injuries

- Etiology: 1- Penetrating chest wounds (More common)
- 2- Compression trauma of abdomen → suddenly raises intra-abdominal pressure → tear in the diaphragm
- Incidence: more common on the **left** side.
- **Commonly missed** at the time of the accident.
- Usually associated with herniation of abdominal viscera into chest
- Presentation: dyspnea, shoulder pain, diminished breath sounds & audible intestinal sounds on the chest.
- Investigations: 1- Plain chest x-rays 2- Chest CT
- Treatment: repair the injury through an abdominal incision



Oesophageal injuries

Etiology

- 1- Iatrogenic: (**the commonest**)
 - During endoscopic dilatation of strictures
- 2- Penetrating or blunt injuries to the neck or chest
- 3- Swallowing of corrosives or F.B.
- 4- Spontaneous perforation
 - Due to forceful vomiting (**Boerhaave's syndrome**)

Clinical picture

- 1- Acute onset of pain at the site of rupture.
- 2- Acute onset of fever, tachycardia and hypotension.
- 3- Mediastinal emphysema
- 4- Pneumothorax and/or pleural effusion.

Investigations

(**Urgent**)

- 1- Plain X-ray → mediastinal emphysema or hydropneumothorax
- 2- Water soluble swallow → reveal the perforation

Management

- 1- Management of shock:
 - I.V. antibiotics and nil by mouth (**Do not pass a N.G. tube**)
- 2- Cervical perforations:
 - a) If early → surgical closure and drainage
 - b) If late → external drainage and parenteral nutrition
- 3- Thoracic perforations:
 - a) If detected early → surgical repair and chest drainage
 - b) If detected late → chest drainage and a feeding jejunostomy then later esophageal reconstruction.
- 4- Abdominal perforation → early surgical repair

Complications

- 1- Mediastinitis
→ rapidly fatal if not urgently treated
- 2- Septic shock



Empyema (pyothorax)

Definition

A collection of pus in the pleural cavity

Acute empyema

Etiology

Route

- 1) 2nd to pulmonary infections: lobar pneumonia (**commonest**), bronchopneumonia, lung abscess or bronchiectasis.
- 2) Contamination from a neighbouring septic focus: ruptures of the esophagus or disrupted oesophageal anastomosis.
- 3) Direct access: open wound (post-traumatic & postoperative)

Causative organisms

- Pneumococci (**commonest**), streptococci and staphylococci.
- Occasionally Gram negative bacilli & anaerobes

Pathological consequences

- **Stage I:** Pre-empyema phase (there is exudation in the pleura)
- **Stage II:** Fibrinopurulent phase
(Bacterial invasion → heavy fibrin deposition & pus formation)
- **Stage III:** Chronic phase (there is massive ingrowth of new capillary loops & fibroblasts → lung is surrounded within a thick layer of fibrous tissue "**fibrothorax**" & cannot expand)

Clinical picture

Symptoms: history of pulmonary infection followed by pain in the chest with dyspnoea, fever and other signs of severe toxæmia.

Signs: of pleural effusion → a) stony dullness
b) Absence of breath sounds c) Tactile fremitus

Investigations

- 1- X-ray → uniform opacity tapering towards the axilla
- 2- Aspiration: confirms the diagnosis & allows organism identification by culture and antibiotic sensitivity tests

Treatment

A) Drainage

- 1- **Pleurocentesis:** introduce the needle one space below the upper border of dullness then aspirate gradually & repeat if pus recollects
- 2- **Intercostal tube drainage:** if pus is too thick or keeps recollecting

B) Antibiotics according to culture and sensitivity.

Tuberculous empyema

- Definition:

Tuberculous infection of the pleura may occur as a complication of rupture a tuberculous cavity.

Treatment:

1. Repeated aspiration
2. Anti-tuberculous treatment.
3. If these fail to allow lung expansion → decortication



Chronic empyema

Definition

Failure of the lung to achieve total expansion (by clinical and chest radiology) > 14 days despite drainage of pleural pus

Etiology

- 1- Inadequate drainage of pus
- 2- Failure to recognize and treat an underlying causative factor(s).

Clinical picture

General:

- 1- General ill-health with anaemia and clubbing of fingers
- 2- Acute exacerbations are common with fever and chest pain

Local:

- 1- Open type → a chronic sinus in chest wall leading to the pleural and discharging pus (continuously or intermittently)
- 2- Closed type → perforates an intercostal space → S.C. abscess (**empyema necessitans**)
- 3- In long-standing cases, progressive fibrosis causes:
 - a) Contraction & rigidity of the chest wall with crowding of ribs
 - b) Elevation of the diaphragm
 - c) Displacement of the mediastinum to the affected side
 - d) Scoliotic deformity of the spine.

Investigations

Laboratory:

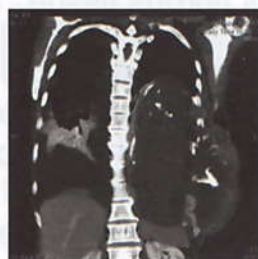
- Bacteriological examination of pus & sputum to exclude TB and to identify the causative organism.

Radiological

- 1- Chest x-ray → may reveal FBs or underlying lung disease
- 2- CT scan of the chest

Treatment

By **decortication** (complete excision of the thickened pleura that forms a tight peel around the lung) → the freed lung expand and obliterate the cavity.



Bronchogenic carcinoma

Etiology

The incidence markedly raised during recent years, because of:

- 1- Excessive cigarette smoking (Both active & passive smoking)
- 2- Inhalation of irritants: as silica, cobalt dust in mines & petrol fumes in large cities.

Pathology

Gross types

1- The hilar type (the commonest 75%)

- Origin: one of the main bronchi or their primary divisions
- Effect: bronchial obstruction with 2^{ry} changes in lung as atelectasis, pneumonia or lung abscess.

2- The peripheral type (25%)

- Origin: the smaller bronchi
- Includes the **Pancoast's** tumour which arises at the apex of lung and invades:
 - A) the brachial plexus
 - B) The first rib
 - C) Sympathetic trunk → **Horner's syndrome**

Histology

A) Non-small cell carcinoma

1- Squamous-cell carcinoma (30-40%)

- Site: Arises centrally
- Advantages: less to metastasizes & most favourable for resection

2- Adenocarcinoma (25-40%)

- Site: arises from peripheral bronchi.
- It is frequently discovered incidentally on chest x-ray
- Chest wall & pleural invasion are dominant

3- Bronchoalveolar carcinoma (5%)

- Site: peripheral
- Spread: to alveoli in other areas of the same or contralateral lung

4- Carcinoid: Low grade, central & has neuroendocrine activity.

B) Small cell carcinoma (20%)

- Site: central
- Usually there is massive mediastinal tumour

Spread

1- Direct: to the mediastinum, pleura, chest wall or pericardium.

2- Lymphatic:

- To hilar LNs then to tracheobronchial & lower deep cervical nodes

3- Blood: mainly to the liver, adrenals, brain and bones

- Bronchial cancer affects both sexes.
- It is commoner in males than females (10: 1)
- Occurs most often between ages of 40 -60 years.
- It has very bad prognosis.

Staging (TNM staging)

Primary tumor	Lymphatic spread	Metastasis
• Tis: Carcinoma in situ • To: No evidence of 1st tumour. • T3: Tumour of any size invading the chest wall • T4: Tumour of any size involving the heart, blood vessels, trachea, esophagus or presence of pleural effusion.	• No: No LN metastases • N1: Metastases to ipsilateral LNs • N2: Metastases to subcarinal or mediastinal LNs	• Mo: No distant metastases. • M1: Distant metastases

• **Stage I:** T₁-T₂ – N₀ – M₀

• **Stage II:** T₁- T₂ – N₁ – M₀

• **Stage III:** T₃–N₀₋₁ or any T- N₃ -M₀

• **Stage IV:** Any T, any N, M1

Clinical picture

1- Insidious type

- Onset is insidious with a dry irritant cough (due to smoking) → then asthmatic & associated with haemoptysis, dyspnea & chest pain

2- Acute type

- Onset is acute with symptoms of pneumonia which fail to resolve satisfactorily or relapses within a few days or weeks.

3- Latent type

- In peripheral carcinoma, the disease may remain silent until:

- Discovered by mass radiography
- Produce vague general symptoms, e.g. fatigue & indigestion
- Distant metastases: ↑intracranial tension (headache, vomiting & blurring of vision), pathological fractures, jaundice & adrenal failure.
- Symptoms of mediastinal invasion (mediastinal syndrome)

4- Pancoast type

- A peripheral tumour at the thoracic outlet (superior sulcus tumour) invades brachial plexus, sympathetic trunk & upper ribs → lower brachial plexus lesion with Horner's syndrome & erosion of upper 2 or 3 ribs

5- Para-neoplastic syndromes

- Undifferentiated tumours may produce hormone like substances, toxins or antigens which produce a variety of manifestations:

- A Cushing-like syndrome due to ACTH production.
- Water retention due to secretion of ADH → hyponatraemia, water intoxication and cerebral symptoms.
- Hypercalcaemia due to a parathormone-like polypeptide.
- Tender gynaecomastia due to ectopic gonadotrophin secretion.
- Carcinoid syndrome due to secretion of 5- hydroxytryptamine or 5-hydroxy tryptophan
- Malignant neuromyopathy which may cause myasthenia, dermatomyositis or peripheral neuritis.
- Pulmonary osteoarthropathy → clubbing of fingers

Pulmonary metastases

- Origin:

From any malignant tumour but are especially common in renal cell carcinoma, melanoma, osteosarcoma & carcinoma of the thyroid, breast, stomach and prostate

- Gross appearance:

Small multiple and bilateral but sometimes large and rounded (cannonball metastases)

Investigations

Radiological

1- Plain chest x-ray: **coin shadow** or hilar opacity or signs of obstructive emphysema, atelectasis, pneumonia, lung abscess, enlarged mediastinal glands, pleural effusion, erosion of ribs or diaphragmatic paralysis

2- CT scan:

- Localizes the tumour & detects mediastinal LN enlargement
- Provides a high accuracy for guidance of needle biopsy in peripheral lesions

Instrumental

1- **Bronchoscopy** (establishes diagnosis & assesses operability)

- In **60%** → tumour is visible as a nodular necrotic mass or an irregular stenosis and a punch biopsy can be taken.

- **Signs of inoperability:**

- a) Left recurrent laryngeal paralysis
- b) Compression of trachea or widening of carina by enlarged glands
- c) Fixity of affected bronchus or malignant infiltration beyond the proximal centimeter of the main bronchus.

2- **Scalene node biopsy** → confirm diagnosis & assess operability.

- The pad of fat over scalenus anterior with contained LNs is removed and examined histologically for malignant infiltration.

3- **Needle biopsy of a peripheral lesion under radiography**

- Will reveal malignant cells in about **60%** of cases.

4- **Mediastinoscopy** → to biopsy nodes of the paratracheal group

Laboratory

- **Sputum cytology** → presence of malignant cells

Bronchogenic carcinoma should be suspected in all patients **> 40** who develop prolonged cough, unresolved or recurrent pneumonia, chest pain or haemoptysis.
- Even when pneumonia responds to ordinary medical treatment, the chest should be x-rayed **3 weeks** after cure

Treatment

A) Non-small cell carcinoma

- 1- Radical resection in operable cases (**stage I and II**)
 - a) Pneumonectomy with removal of mediastinal nodes (the best)
 - b) Lobectomy preferred in elderly patients with peripheral carcinoma
- 2- In early stage **III** → neoadjuvant chemo-radiotherapy may render the case resectable.
- 3- Radiotherapy is useful in relieving:
 - a) SVC obstruction
 - b) Bronchial obstruction
 - c) Pain of skeletal metastases.

B) Small-cell carcinoma → Chemo-radiotherapy



Postoperative pulmonary complications

Predisposing factors

A) Preoperative

- 1- Age of the patient (Extremes of age)
- 2- Sex → Males are more predisposed than females
- 3- Smoking: Heavy smokers commonly have chronic bronchitis.
- 4- Chronic bronchitis → excessive production of mucous & damage of cilia → stagnation of mucus in the bronchial tree
- 5- Dehydration → thick bronchial mucus & difficult to expel

B) Operative and post-operative

1- Anaesthetics:

- I) Trauma to the tracheobronchial tree
- II) Premedication with atropine
- III) Prolonged unconsciousness

Predispose to
respiratory problems

- 2- Nature of the operation: wound pain in thoracic & upper abdominal operations → interferes with deep breathing & coughing
- 3- Abdominal distension (due to paralytic ileus) → interferes with the movement of diaphragm and reduces the vital capacity.
- 4- Postoperative pain: interferes with deep breathing & coughing → stagnation of secretions.
- 5- Excess use of narcotics → interferes with breathing & coughing

Types of complications

- | | |
|---------------------|--------------------------|
| 1- Atelectasis | 2- Pulmonary embolism |
| 3- Bronchitis | 4- Mendelson's syndrome |
| 5- Bronchopneumonia | 6- ARDS |
| 7- Lung abscess | 8- Postoperative hypoxia |

Postoperative atelectasis (pulmonary collapse)

Predisposing factors

- 1- Inhalation of FB or vomiting due to full stomach.
- 2- Production of tenacious mucus because of:
 - a) Pre-operative respiratory tract infection
 - b) Premedication with atropine
 - c) Postoperative dehydration
- 3- Inhibition of the cough reflex due to:
 - a) Old age and debility
 - b) Pain
 - c) Excessive sedation particularly with morphine.
 - d) Operations on the chest, diaphragm or upper abdomen.
 - e) Lack of mobility in bed
 - f) Morbid obesity
- g) Recent stroke

Pathology

- Obstruction of a bronchus by a plug of mucous → absorption of air distal to obstruction & deflation of the affected area.

-The consequent collapse may be:

- **Lobular:** collapse of scattered areas throughout the lung.
- **Lobar:** collapse of one lobe usually the lower.
- **Massive:** collapse of the whole lung.

Clinical picture

Symptoms

- 1- May manifest within **48 hours** after operation or pass unnoticed until complicated by serious lung infection.
- 2- In typical cases, the onset is acute with fever, dyspnoea, cyanosis & chest pain in massive cases
- 3- Cough is slight & sputum is scanty and difficult to expel.
- 4- May subside within a few days with the expectoration of a considerable amount of purulent tenacious mucous

Signs

- 1- A minor atelectasis produces no local signs.
- 2- Unexplained tachycardia.
- 3- With collapse of a segment or whole lung → the affected side is flattened & immobile with dullness on percussion, diminished breath sound and displacement of the trachea and heart towards the side of collapse
- 4- As the attack subsides → tubular breathing & coarse crepitations

Investigation

X-ray shows:

- a) The collapsed lobe → homogeneous wedge-shaped opacity.
- b) Major atelectasis →
 - i) Approximation of the ribs
 - ii) Elevation of the diaphragm
 - iii) Deviation of the mediastinum toward affected Side

Prophylaxis

• Preoperative

- 1- Eradication of respiratory infections
- 2- Stop smoking for **6-8 weeks** for elective cases
- 3- Breathing exercises.
- 4- Avoidance of dehydration.
- 5- Avoidance of heavy premedication (particularly with atropine & morphine)

• Operative.

Avoidance of

- 1- Trauma to upper air passages during intubation
- 2- Deep unconsciousness & vomiting under anaesthesia
- 3- Tight strapping and bandaging of the chest & abdomen

Atelectasis is the precursor of majority of post-operative chest complications, particularly pneumonia & lung abscess



• Post-operative

- 1- Correction of dehydration.
- 2- Avoidance of heavy sedation
- 3- Encouragement of deep breathing, & postural exercises
- 4- Removal of secretions by coughing, expectoration & postural drainage.
- 5- Early ambulation
- 6- Adequate analgesia to reduce pain with coughing & deep breathing

Treatment

1- Try to expel the obstructing plug by:

- Turning the patient from side to side.
- Percussing the affected side of the chest.
- Encouraging expectoration and breathing exercise.
- Loosening sputum by mucolytics, expectorants & steam inhalation
- Suction

2- Antibiotics → to prevent infection.

3- Oxygenation

- If O_2 saturation & PO_2 are low → intubate with mechanical ventilation

If aeration does not occur within **4-6 hours** → remove the bronchial plug by bronchoscopic suction

Bronchopneumonia

-Etiology: sequel to bronchitis & atelectasis.

-Causative organisms: Haemophilus influenza, pneumococci, staphylococci or Pseudomonas pyocyaneus.

-Pathology: Multiple areas of patchy consolidation

- Presentation: profound toxemia

- Investigation: 1- Chest X-ray → multiple areas of patchy mottling

2- Sputum culture → reveals the responsible organisms.

- Prevention: the same as prophylaxis for collapse.

-Treatment:

- Physiotherapy and antibiotics.
- Bronchoscopic removal of secretions may be required.

Mendelson's syndrome

• Etiology: aspiration of vomitus which contains irritant HCL (During induction of anaesthesia in patient with full stomach or I.O. or in pregnancy & in comatose patients after head injury or drug poisoning)

• Presentation: wheezes, cyanosis, tachycardia, tachypnea and hypotension

• Radiologically: widespread lung infiltration more on right than on left and more in the lower lobes

• Laboratory: ABG → severe hypoxia.

• For prophylaxis, all high-risk patients should have a NG tube inserted before the operation for suction of the gastric contents.

• Treatment: a) Bronchoscopic aspiration

- Steroids
- Bronchodilators
- Antibiotics

Bronchitis

-Incidence: the commonest postoperative complication → predispose to severe complications as burst abdomen.
- It may occur de novo or represents exacerbation of a pre-existing bronchitis.

-C/P:

- Post-operative cough with mucopurulent sputum
- Severe suppurative bronchitis

-Investigation: Chest x-ray → free

Adult respiratory distress syndrome (ARDS, shock lung)

Etiology

- 1- Severe sepsis
- 2- Major trauma
- 3- Major burns
- 4- Severe acute pancreatitis
- 5- Severe shock from any cause especially if requiring large volumes of intravenous fluids (particularly in septic shock)
- 6- Lung injury due to trauma, inhalation of fumes or aspiration of gastric contents

Pathogenesis

Activation of platelets, neutrophils and macrophages → release of oxygen free radicals → causing:

- 1- Endothelial damage
- 2- Oedema of lung tissue
- 3- Increased capillary permeability → neutrophils & RBCs move to the inside of alveoli

- Defects occur in the three aspects of the respiratory process:

1- Defective ventilation

- The loss of surfactant reduces lung compliance and expansion
- Alveolar edema (presence of fluid in the alveoli) prevents air from reaching the gas exchange surface

2- Defective perfusion

- Lung perfusion is affected if the cause of ARDS is shock
- Deoxygenated blood is shunted directly from some of the arterioles to the venular sides through opening of A-V shunts.

3- Defective diffusion

- The distance between the alveolus and the capillary (known as **alveolocapillary membrane**) is thickened because of the interstitial pulmonary edema
- O_2 & CO_2 find difficulty in crossing this thickened membrane.

Clinical progress

1- Initial state of shock

- Lactic acidosis & hyperventilation with low $PaCO_2$

2- Phase of hemodynamic equilibrium (last several days)

- The patient may appear well & recovering with a normal chest x-ray but the PaO_2 is invariably low

3- Phase of clinical respiratory distress

- Followed by respiratory failure with confusion and occasional Petechial rash.
- Rising $PaCO_2$ & falling PaO_2 occur despite O_2 supplement.
- Chest x-ray reveals bilateral pulmonary infiltrations

Pathology

A) Macroscopic picture

- 1- Great increase in lung weight
- 2- Petechial hemorrhages on epithelial surfaces.

B) Microscopic picture

- 1- Interstitial edema & haemorrhage
- 2- Alveolar edema
- 3- Perialveolar haemorrhage.
- 4- Intravascular fat globules & fibrin plugs



Treatment

The patient should be admitted to ICU

1- Respiratory support

- In stage of respiratory distress, an oxygen mask is enough to improve arterial blood oxygen
- In stage of respiratory failure (indicated by a $\text{PaO}_2 < 60$ mmHg) endotracheal intubation and mechanical ventilation are required.

2- Treatment of the cause as correction of shock & eradication of sepsis

Postoperative hypoxia

Common causes

- 1- Pulmonary aspiration
- 2- Failure to breathe deeply & cough during anesthesia recovery
- 3- Airway blockade by secretions → alveolar collapse
- 4- Hypoventilation due to pain of upper abdominal or thoracoabdominal incisions, opiates overdose, anesthetics or prolonged recumbency
- 5- Pulmonary embolism

Clinical picture

- | | |
|---|---------------------|
| 1- Restlessness, anxiety or confusion | 2- Tachypnea |
| 3- Tachycardia, dysrhythmias or hypotension | 4- Central cyanosis |

Investigations

1- Pulse oximetry

Probe attached to 1 finger & shows O_2 saturation (normal **95-100%**)

2- Arterial blood gases

- Increased PCO_2 denotes ventilation failure & decreased O_2 denotes failure of any of the three components of respiration

3- Chest x-ray

- Reveals underlying cause (as collapse or pneumothorax)

Treatment of post operative hypoxia:

- 1- Treat the specific cause
- 2- If respiratory failure occurs
→ Mechanical ventilation

Mediastinal tumours

Pathology

1- Tumours of the anterior & superior mediastinum include:

Retrosternal goitre, thymic tumours, dermoid cysts & teratomas

2- Tumours of the posterior mediastinum may be

- a) Cysts of congenital origin, e.g., bronchogenic, gastrogenic and lymphatic cysts
- b) Lymphadenopathy of secondary deposits, TB, lymphoma or leukaemia.
- c) Solid tumours of connective tissue or neurogenic origin, e.g., neurofibroma, ganglioneuroma and lipoma.

Clinical picture

Mediastinal syndrome

- 1- Dyspnoea (due to pressure on the air passages or encroachment on the intrathoracic space)
- 2- Dysphagia from compression of the oesophagus.
- 3- Oedema, cyanosis and dilated veins in head, neck & upper limbs (Form obstruction of the SVC)
- 4- Hoarseness of voice & brassy cough (due to involvement of recurrent laryngeal nerve)
- 5- Hiccup or diaphragmatic paralysis (from phrenic N. involvement)
- 6- Arrhythmias & EGG changes (If there is pericardium invasion)

Diagnosis

- 1- Plain X-ray → opaque shadow in the mediastinum
 - Screening reveals the expansile pulsation of an aortic aneurysm or movement of retrosternal goitre during swallowing
- 2- CT scan → localize the site and origin of the tumour.
- 3- Bronchoscopy → to exclude bronchogenic tumours.
- 4- Mediastinoscopy → to take a biopsy

Treatment

- 1- Radiotherapy: for enlarged malignant LNs & malignant tumours and may be supplemented with chemotherapy.
- 2- Excision: for cysts & simple tumors

Cardiac operations

Preoperative diagnostic procedure

1- ECG

a) Resting: Rhythm, conduction abnormalities, chamber hypertrophy, established ischaemic changes & old M.I.

B) Exercise: Exercise-induced ischaemic changes.

2- **Chest x-ray**: Cardiac enlargement & valve calcification

3- **Thallium isotope scan**:

Low isotope uptake indicates impaired myocardial perfusion.

4- **Echocardiography**: Myocardial contractility, valve stenosis or regurgitation & septal defects

5- **Cardiac catheter**

a) Chamber pressures: Assess left & right ventricular functions & assess valve diseases.

b) Angiography: Coronary artery anatomy.

c) O₂ saturation (Intracardiac shunts)

d) Cardiac output: Cardiac function & pulmonary vascular resistance

Thymic tumours

-Incidence: rare.

- Include: thymoma (epithelioma), lymphomas, teratoma and lymphangioma

-Presentation:

1. May be symptomless & discovered accidentally on X-ray examination,
2. May present with features of myasthenia gravis or features of mediastinal syndrome.

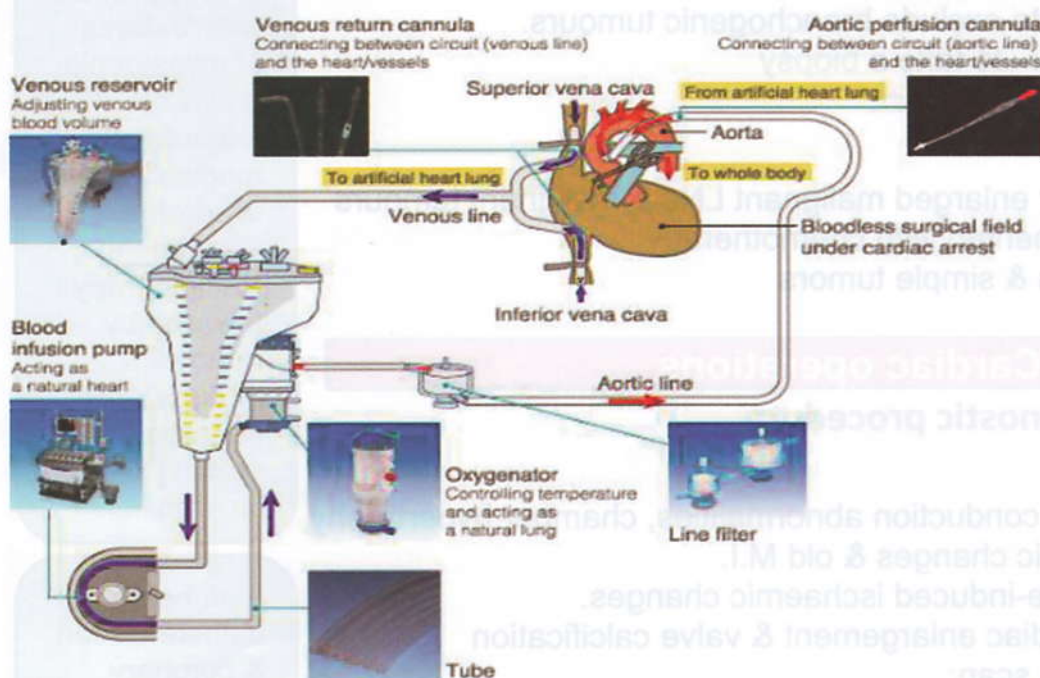
-Treatment: Radiotherapy followed by removal through a sternum splitting incision

Left heart catheterization & coronary angiography are done by inserting a fine catheter into brachial or femoral artery & threading it up to aorta
- Similarly, right heart catheterization is performed through a peripheral vein

Operative procedure

Extracorporeal circulation (the pump)

- Most cardiac operations are performed under direct vision in a bloodless field within the chambers of the heart or great vessels.
- A median sternotomy is used
- The venous inflow to the heart is diverted through an artificial heart lung machine (extracorporeal circulation) which consists of:
 - 1- Roller pumps (propel blood to and from the heart)
 - 2- An oxygenator (supplies oxygen and removes CO₂)
 - 3- Heat exchanger that controls the blood temperature.
- The machine is filled with fresh heparinized blood
- Venous blood is collected via cannulae inserted into SVC & IVC
- Venous blood is pumped through the oxygenator and heat exchanger and is returned as arterial blood by a cannula into femoral artery or aorta.



Cardiac surgery complications

1- Complications of extracorporeal circulation

- A) Cerebral ischaemia (either severe → infarction or minimal → transient postoperative psychological or memory disturbances)
 - b) Activation of cytokines that induce SIRS (usually minor but in rare cases may cause coagulopathy and renal failure)
 - c) Haemolysis.
- 2- Low cardiac output
 - 3- Arrhythmias
 - 4- Pulmonary infection.
 - 5- Mortality (from 2% for routine straightforward procedure to > 50% for complex emergency procedures)

During extracorporeal circulation, the heart is not perfused. The myocardium is usually preserved by a combination of

1. Cardioplegia

The heart is induced to stop pulsations by infusing a cardioplegic solution in the coronary arteries (has high potassium content) → minimizes myocardial oxygen requirement & allows surgeon to operate at comfort on a non-beating heart

2. Systemic hypothermia

down to 32°C reduces myocardial need for O₂

Cardiac arrest

Definition

- Cessation of effective cardiac output as a result of ventricular asystole, ventricular tachycardia, or ventricular fibrillation (VT/VF)
- The end result is sudden cardiac death

Etiology

1- Sudden severe reduction of venous return to the heart

- a) Sudden severe haemorrhage
- b) Failure of cardiac filling (due to pericardial tamponade, massive pulmonary embolism or tension pneumothorax)
- c) Severe peripheral VD (may occur after spinal anesthesia)

2- Severe depression of the myocardial function

- a) Massive M.I.
- b) Hypoxia or hypercarbia
- c) Irritating drugs as epinephrine
- d) Severe traumatic myocardial contusions.
- e) Acidosis & Hyperkalaemia
- f) Hypothermia

Types

According to the ECG findings

1- Shockable rhythm: ventricular tachycardia or fibrillation

2- Non-shockable rhythm: cardiac asystole & pulseless electrical activity (there is ECG rhythm compatible with a pulse but pulse is absent)

Diagnosis

The cardinal signs are: 1- Absent carotid pulse.

2- Absent or gasping respiration (look, listen and feel for air).

3- Dilated pupils.

4- Under anesthesia → ECG monitoring of the arrest & sudden cessation of bleeding (main features)

Management

- The brain can only tolerate **3 minutes** of complete circulatory arrest & unless circulation is restored within this period → death or permanent neurological damage if the patient survives.

-Management is started **immediately** and consists of three phases.

1-Pre-hospital management (CPR)

• External CPR by closed cardiac massage and direct mouth-to-mouth breathing

• Aim: not to restart the heart but to provide an artificial circulation and artificial ventilation until appropriate facilities are available

The common causes of cardiac arrest are:

• 5Hs:

Hypovolaemia,
Hypoxia,
Hyperkalaemia
Hydrogen ion excess &
Hypothermia

• 5Ts.

Thrombosis
(coronary or pulmonary),
Tension
pneumothorax,
cardiac
Tamponade,
Toxic orders
and Trauma

Stethoscope has no place in diagnosis of cardiac arrest. Once suspected & the carotid pulse is not palpable → start resuscitation immediately

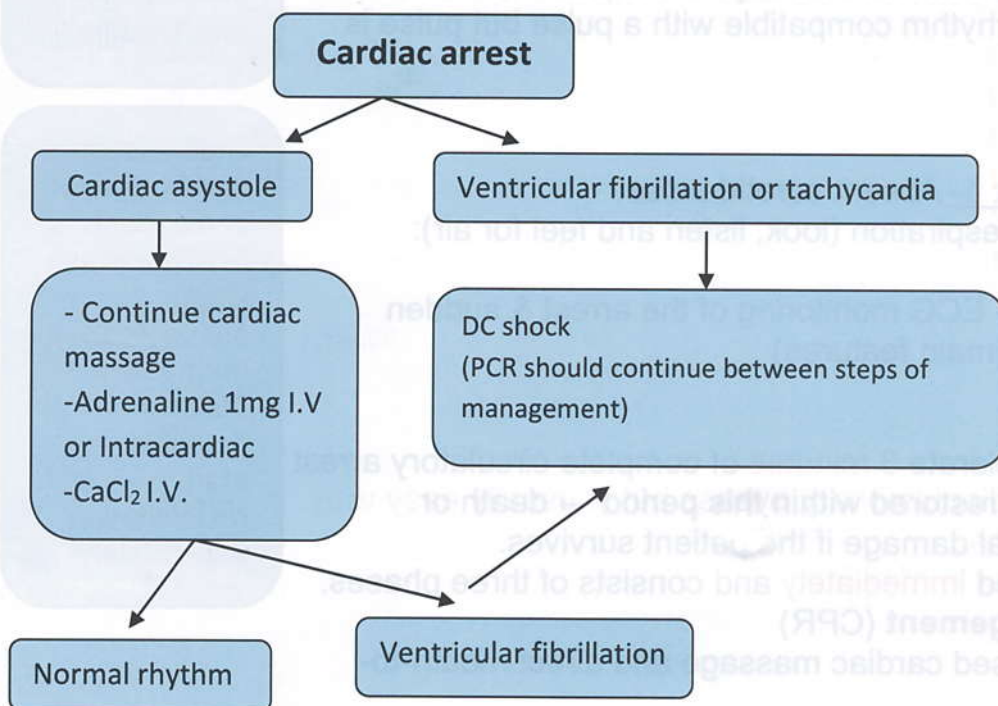
• **Technique:**

- a) Patient is laid flat on a firm surface & legs are raised to ↑VR
- The person who performs external cardiac massage should press lower $\frac{1}{2}$ of sternum by heel of both hands, the elbow should be straight & chest is compressed 4-cm at a rate of 100/min
- b) A second person performs mouth to mouth breathing.
- The head is tilted back with one hand to ensure a clear airway & the nostrils are closed with other hand while mouth is applied to patient's mouth to fill his lungs with expired air at a rate of 10-15 times /min
- Use of a special (tube & bag) is preferable to avoid cross-infection
- c) The efficiency of ventilation and massage must be monitored by observing chest expansion & palpating femoral pulse
- d) Patient should then be transferred to hospital as soon as possible, with direct mouth-to-mouth breathing & cardiac massage continued until endotracheal intubation & medical treatment are available

2- Hospital management

• **Aim:** restore normal cardiac rhythm

- An endotracheal tube is passed & lungs are ventilated with O₂
- A venous cannula is inserted and suitable infusions are given.
- ECG monitoring is applied to differentiate cardiac asystole from ventricular fibrillation or tachycardia.
- Further management depends on ECG diagnosis



Open cardiac massage

- **Indication:**

rarely if chest is already opened as during a chest operation or if closed massage has been unsuccessful.

- **Technique:**

The chest is opened through the left 5th intercostal space & the pericardium is widely opened to permit bimanual massage with one hand above & other below the ventricular mass

- **Rate:** 60-80/minute

- **Duration:**

Until the cardiac tone returns & heart becomes smaller, firmer and pinker

3-Subsequent treatment (in ICU)

- Measures are taken to prevent and to treat re-arrest:

- 1- Blood pressure kept **> 90 mmHg** by vasopressors & transfusion
- 2- If chest was opened it is left open for ½ an hour in case re-arrest
- 3- Cardiac stimulants, e.g. epinephrine, calcium chloride & digitalis may be administered as needed.
- 4- Hypothermia & dehydration therapy with mannitol or concentrated glucose are induced if return of consciousness is delayed.
- 5- Infusions of NaHCO_3 solution are given to correct the metabolic acidosis produced by anoxia & blood transfusion
- 6- Hyperkalaemia is often present and should be treated with CaCl_2 or glucose & insulin
- 7- Anuria and renal damage should be anticipated and prevented
- 8- The cause of cardiac arrest must be investigated and treated

Principles of surgery for congenital heart diseases

- Timing of Surgery: early after diagnosis (particularly for cyanotic heart disease to prevent development of irreversible lung changes that lead to pulmonary hypertension)

Patent ductus arteriosus

-Treatment: surgical closure

-Timing: best between the ages of **3 -5 years**

-Operation: Through a posterolateral thoracotomy the ductus is exposed & ligated using nonabsorbable material (no need for cardiopulmonary bypass)

Coarctation of aorta

-One of the surgically-correctable hypertension.

-Timing: between the ages of **5 -15 years**

-Treatment:

- a) Excision of stenosed part & end-to-end anastomosis, if possible.
- b) Excision & grafting with Dacron prosthesis (in long constriction)

Atrial and ventricular septal defects

- Type of patient: either in children or adults.

- Surgery requires cardiopulmonary bypass but is usually easy with low risk of complications.

- Repair: according to size of defect

- a) Small defects → closed directly by sutures
- b) Large defects → closed by Dacron grafts

Key points in management of cardiac arrest

- CPR should continue between the steps of management.
- If there is doubt whether the rhythm is asystole or fine VF → does not attempt defibrillation instead continue good quality CPR which may improve the amplitude & frequency of VF & improve chance of subsequent successful defibrillation

Surgically correctable hypertension

- 1- Renal diseases
- 2- Renal artery stenosis
- 3- Polycystic kidneys
- 4- Coarctation of aorta
- 5- Endocrine diseases
- 6- Pheochromocytoma
- 7- Conn's syndrome
- 8- Cushing's syndrome

Tetralogy of Fallot

- Consisting of four defects:

- 1- Stenosis of the pulmonary artery.
- 2- Interventricular septal defect.
- 3- Dextroposition of the aorta which overrides the septal defect & receives blood from both ventricles
- 4- Hypertrophy of the right ventricle.

- Cyanosis is marked since birth → **blue babies**

- Treatment:

1- **Blalock's operation** (Temporary palliative shunt).

- The subclavian artery is anastomosed to the pulmonary artery → improves blood flow to the lungs

- Done in 1st year or two of life if cyanosis is severe enough to make total correction with cardiopulmonary bypass dangerous

2- **Total correction** (the definitive procedure)

- It requires cardiopulmonary bypass.

- The operation is usually advised before school age.

Principles of surgery for acquired valvular heart diseases

Mitral stenosis

- Indications for surgery:

- 1- Progressive limitation of exercise tolerance
- 2- Nocturnal dyspnea
- 3- Atrial fibrillation
- 4- Congestive heart failure.
- 5- Mitral valve opening $< 1 \text{ cm}^2$ as calculated by echocardiography

- Optimal timing: between 20 -50 years

(< 20 → reactivations of rheumatic disease may lead to recurrence, while > 50 → myocardial ischaemia, atherosclerosis or emphysema)

- Methods:

1-Mitral valvotomy

- Prerequisite: no associated incompetence & valve is not calcified
- Method: by a percutaneous balloon or by open heart surgery.

2-Mitral valve replacement

- Prerequisite: patients with mixed stenosis and incompetence & for calcified valves

Mitral incompetence

• If the valve is not heavily distorted or calcified → valve repair is preferred than replacement (narrow the valve annulus just enough to induce competence)

• If there is associated stenosis or heavily distorted and calcified → Mitral valve replacement

Aortic valve disease

- Treatment: Valve replacement on cardiopulmonary bypass
(Associated coronary artery disease is treated at the same time)

- Types of artificial valves:

1- Prosthetic valves:

- Have different designs as caged ball & tilting leaflets.

- Turbulent flow through these valves → produce thrombosis →

Patients take anticoagulants for life

2-Bio-prosthetic valves

- Valve leaflets are porcine, bovine or human from fresh cadavers.

- Valve is suspended on a prosthetic ring to allow it to be sewn

- The recipient does not need anticoagulation.

- The lifespan of such valves is shorter than prosthetic valves but anticoagulation avoidance makes them preferred choice in elderly.

- Complications of valve replacement:

1- Prosthetic valve thrombosis (if anticoagulants are discontinued)

2- Valve breakdown.

3-Complications of anticoagulants (particularly bleeding)

4- Valve infection (similar to bacterial endocarditis of native valves)



Ball and cage



Tilting disc



Bioprosthetic or tissue valves

Ischaemic heart disease

Etiology

Narrowing of the coronary arteries by atherosclerosis

Pathology and diagnosis

- The most common symptom: angina pectoris, but the disease may end in coronary thrombosis with M.I, heart failure or ventricular fibrillation and sudden death

-Diagnosis: by the history, ECG & exercise tolerance tests.

(Coronary arteriography is necessary before surgical treatment to determine occlusion sites & patency of vessels beyond obstruction)

-In atherosclerosis, the basic lesion is a segmental atheromatous plaque (often within 1st 5 cm of the origin of a coronary artery)

- Multiple areas of occlusion are common & about **50%** of patients with severe angina have involvement of all 3 major arteries (anterior descending, circumflex & right coronary)

Treatment

A- Medical

1- Moderation of activity

2- Control of risk factors: Prohibition of smoking & control of DM, hypertension & hyperlipidaemia

3- Medications: Coronary vasodilators (nitrates), beta blockers and calcium channel blockers

B) Coronary revascularization

-Indications: Severe ischaemia despite adequate conservative ttt

-Methods:

1- Percutaneous transluminal coronary angioplasty (PTCA)

Indication: for a short arterial narrowing

Technique: by inserting a balloon catheter inside stenosed artery. The balloon is inflated to dilate the artery then the dilated artery is usually supported by a stent to keep it open.

2- Coronary artery bypass graft (CABG).

o Indications:

- i) Coronary arteries those are not suitable for PTCA
 - ii) Stenosis of main stem of left coronary artery.
 - iii) Three-vessel disease, particularly in diabetics.
 - iv) Surgery for complications of infarction (include mitral valve reflux due to rupture of papillary muscle, ventricular septal defect due to damage of septum & aneurysm of left ventricle due to its weakness)
- Operative correction of these complications is commonly associated with coronary bypass & mortality is high

o Technique

- i) Open cardiac operation using extracorporeal circulation & cardioplegia
 - ii) Coronary bypass can be done by either:
 - 1- Reversed saphenous vein grafts (anastomosed proximally to ascending aorta & distally to diseased coronary artery beyond the stenosis)
 - 2- Internal mammary artery (employed as a pedicle graft, when it is left attached to subclavian artery proximally & to the diseased coronary artery distal to stenosis)
 - Has better **5&10 year** patency rates than saphenous vein grafts
 - 3- A combination of both types is usually employed.
- The left internal mammary artery is used for a vessel at the front of the heart & vein grafts are used to bridge other occlusions
- 4- Recently the radial artery is used as a vascular graft

o Results of CABG:

- 1- Angina relieved completely in **70%** & improved in the remainder
- 2- Angina recurs with a frequency of about **10% per year**.
- 3- Carries a mortality of **2-3%** and **1-2%** risk of stroke

o High risk factors for morbidity or mortality for CABG surgery

- 1- Advanced age **>65** years
- 2- Female sex (thinner fragile coronaries).
- 3- Serious lesions affecting main trunk or multiple coronaries.
- 4- Stenosis affecting left main coronary artery.
- 5- Poor coronary distal run –off
- 6- Dilated heart
- 7- Left ventricular ejection fraction **<30%**.
- 8- Recent M.I. (before **6 weeks**)
- 9- Associated valvular lesions.
- 10- Preoperatively uncontrolled systemic disease

Coronary anatomy

- There are 2 main coronary arteries (right & left coronaries)
- Origin: Both from ascending aorta
- Left coronary artery divides into:
 1. LAD coronary → supplies the anterior wall of left ventricle & anterior 2/3 of interventricular septum
 2. Circumflex coronary → supplies the posterior & lateral walls of left ventricle.
- The right coronary artery supplies right atrium & right ventricle (through its marginal branch)
- The RCA, LAD and CX arteries each considered to be a "**vessel system**" → disease of any one of them or its branches is termed one vessel disease.

Thoracic aortic aneurysms

- **Etiology:** 1- Atherosclerosis (commonest) 2- Trauma
- 3- Marfan syndrome. 4- Syphilitic aneurysms (rare)
- **Presentation:** (mediastinal syndrome)
 - It compresses SVC, trachea, esophagus & recurrent laryngeal nerve
 - It may also erode the vertebrae.
- **Investigations:** CT scan or MRI
- **Treatment:** Surgical excision of diseased segment & interposing a synthetic graft (3 branches of the arch are implanted in the graft)
- **Complication surgery:** paraplegia due to interrupting the blood supply of the spinal cord.

• Aneurysmal dilatation may affect the aortic arch, the descending thoracic aorta or may extend into the abdomen forming a thoraco-abdominal aortic aneurysm

Aortic dissection

Predisposing factors Atherosclerosis and hypertension

Pathology

- An intimal tear → blood dissect its way in the media → false lumen → rupture (inside true lumen or to the outside) → fatal haemorrhage
- As blood dissects its way distally → occludes ostia of aortic branches → ischaemia (of particular importance are the coronaries, the renal & mesenteric arteries)

Types

Type A (more serious)

- Dissection starts in ascending aorta → aortic incompetence, coronary artery occlusion & fatal cardiac tamponade

Type B: Dissection starts distal to the arch

Clinical picture

- Severe chest pain that radiates to the interscapular region.
- It is commonly misdiagnosed as acute myocardial infarction (MI)

Investigations

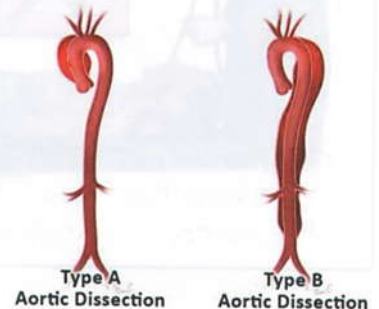
- 1- ECG
- 2- CT
- 3- Aortography
- 4- Echocardiography (particularly trans-oesophageal)

Treatment

1-Type A → **urgent surgery** for the placement of a synthetic graft at the root, replacement of aortic valve and reimplantation of the coronaries in the graft

2-Type B → **antihypertensive.**

- Elective surgery may be needed in chronic extension of dissection.



MI may also be present in type A & diagnosis is not easy

Bronchoscopy

Indications

Diagnostic

In bronchogenic carcinoma (see a central lesion & take biopsy)

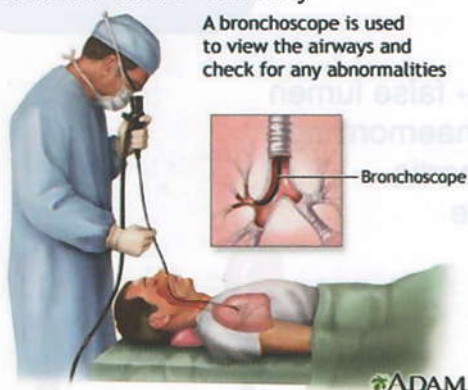
Therapeutic

- 1- Removal of FB
- 2- Dilatation of airway stricture
- 3- Bronchial suction of secretions in cases of resistant atelectasis.
- 4- Laser ablation of obstructing bronchogenic carcinoma.

Types

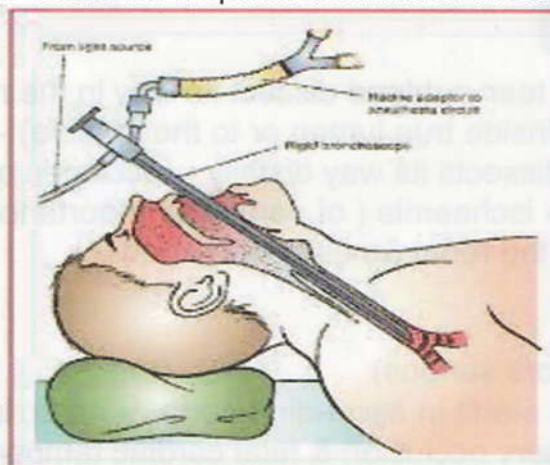
Fibreoptic bronchoscopy

1. Does not necessitate general anaesthesia
2. More convenient
3. Allows wider visibility



Rigid bronchoscopy

1. Requires general anaesthesia
2. Better for removal of foreign bodies & secretions
3. Better for biopsies & control of bleeding



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Surgitoons